Martin Elton

EVENT-RELATED ELECTROCORTICAL RESPONSES ASSOCIATED WITH CHANGES IN MOTIVATION IN REACTIVE DEPRESSIVES

Dissertation
zur
Erlangung des akademischen Grades
Doktor der Sozialwissenschaft
in der Fakultät
für Sozial- und Verhaltenswissenschaften
der Eberhard-Karls-Universität Tübingen

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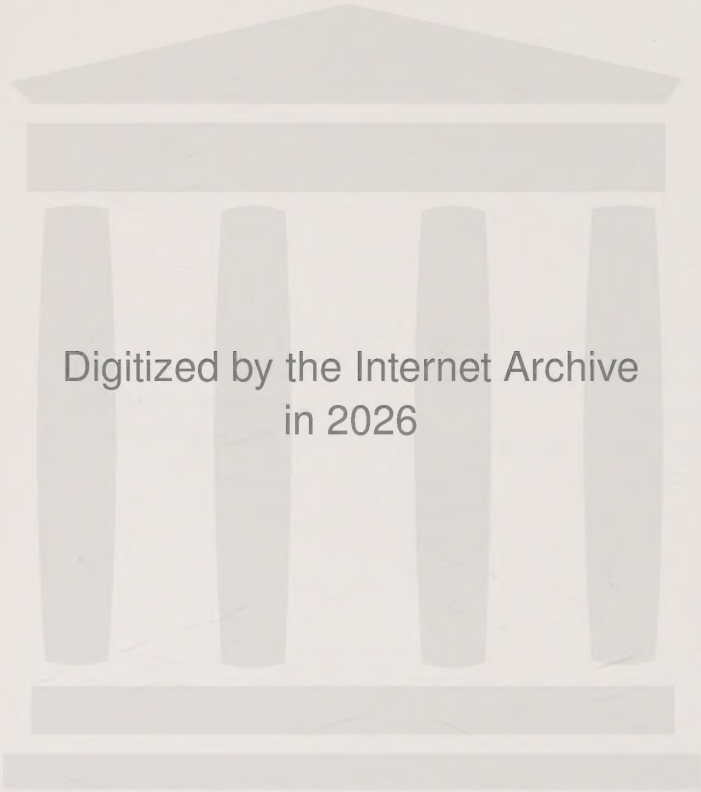
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EVENT-RELATED POTENTIALS.

1. Introduction.

The following section is concerned with presenting a few comments on the nature, from physiological and psychological standpoints, of event-related potentials. Such is not intended to be exhaustive, merely introductory. Lack of success in establishing definite relationships between event-related potentials and such psychological processes as attention, arousal, and motivation has led some researchers to question the feasability of the contemporary research orientation ever providing such solutions. Detailed discussion will focus on the ideational maturation of the Contingent Negative Variation (CNV) and the P300. Further information, of particular relevance to recording in the clinical situation, is introduced in a later section.

The study of stimulus-response contingencies, as reflected in the evoked response, affords insight into the nature of information processing mechanisms within the organism (Vaughan and Ritter 1973); this term being synonymous with 'complex psychological processes' (Donchin 1977). The averaged evoked potentials (EP) have been divided into four groups (Vaughan 1969; Vaughan and Ritter 1973); these being stimulus evoked potentials, association cortex, or long latency, potentials, motor potentials, and slow potential shifts. Figure 1 depicts a stylised diagram of EP components. The EP is a series of electrical changes recorded in temporal association with a physical or mental event (Picton et al. 1978). Variations in early EPs across subjects tend to be more marked in compound amplitude rather than latency, so that a standard sequence may generally be identified for a given experimental condition.

In the first 10 msec, of the auditory evoked potential depicted here, a characteristic waveform is observed which, by its consistency and latency, indicates transmission along

the auditory pathway from the cochlea to the inferior collicus. These early, or 'far-field potentials', are of value in clinical neurology. Following these, in the 50 msec

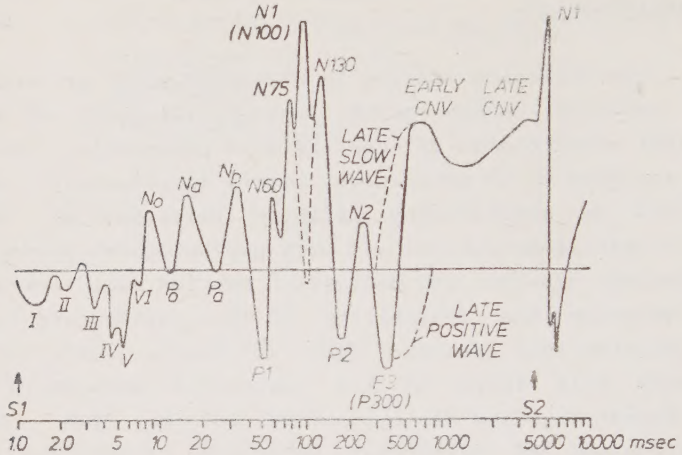

Figure 1. Diagramatic outline of evoked-potential components to an auditory stimulus. Letter prefixes denote polarity. Components do not necessarily occur at these latencies, nor are all seen in every record without special averaging techniques. (From: McCallum 1980).

after stimulation, are other components whose origin is less certain but which follow the arrival of the sensory signal at the primary cortical receiving area, and may have their origin there, in the thalamus, or in the association cortex. The later components, principally the N1-P2-N2 complex, were initially taken as a non-specific response of the frontal association cortex to incoming sensory information. More recently it has been suggested that this response may constitute multiple subcomponent processes, modified by such psychological processes as attention. Finally, the P300 is taken as cortical in origin, but may be elicited in the absence of a physical stimulus. The slow wave, positive posteriorly and negative anteriorly, is almost always assoc-

iated with the late positive wave but having a much longer duration (Vaughan and Ritter 1973; Picton et al. 1974; Donchin 1977; McCallum 1980; Picton and Stuss 1980). McCallum (1979) lists four types of slow potential change; namely the Contingent Negatatve Variation, the Bereitschaftspotential (BP), infra-slow cortical changes, and very slow, or tonic, shifts in the so-called cortical steady states. The CNV, BP, and P300 are refered to as 'event-related slow potentials' (ERSP) often.

A simple division has been made between early and late potentials leaving the status of middle potentials unclear, on the basis of their being 'evoked' or 'invoked'. Early potentials are evoked and owe their features to the physical characteristics of the stimulus. By contrast, the late components, particularly the P300 and including the CNV, are invoked in that they involve processes brought about by the psychological demands of the situation (Donchin et al.1978). This is more often expressed as an endogenous-exogenous distinction in which the latter relates to the subject's prior experience, intentions, decisions, and expectations as modulated by the nature, context, and demands of the current task (Donchin 1977; Donchin et al. 1978; McCallum 1979). Shagass (1976) sees these as reflecting stimulus and response set modes of attention respectively. The latter group is not modality specific and is maximally recorded at the vertex.

As yet no single unifying theory has emerged, capable of relating all ERP findings to behaviour. Thus, much of the work is "phenomenological" (McCallum 1980) and its approach "electrophrenerrianological", that is the localisation of psychological processes through the analysis of electrical 'bumps' on the skull (Rebert 1978). Noting that the understanding of slow potentials is being advanced more by basic physiological and molecular neurobiological studies, Rebert (1978, 1980) argues that knowledge of the causal mechanisms of ERPs is critical to their full understanding and to their proper interpretation in relation to behaviour

and covert psychological processes, e.g. cognition or attention. By contrast, Donchin and Isreal (1980) note that there exists a clear and visible relationship between ERP studies and specific issues that develop within the framework of a well-articulated psychological theory. Two contrasting views of information processing activities are evident; each of which dictates a different approach to psychophysiological investigation. The S-R model approach, more typical of Rebert's 'bump' studies, is concerned with activities occuring between the stimulus and response. The response is seen as a function of the stimulus, the subject's task, the content of memory, motivation level, and so on. Particularly damaging to such an approach are ERP components occuring after the behavioural response; that is, after information processing is supposed to have ceased. As examples, the P300 and post-imperative negative variation (PINV) may be suggested. By contrast is the cognitive approach which rather views information processing as a continuous stream against which memories, expectations and strategies are applied. Accordingly, they recognise a shift in emphasis from the concentration on 'faculties' or states, e.g. attention, orienting, or suprise, to the development of models detailing processes underlying cognition and behaviour. Due to the temporal overlap of physiological activity, which can only be identified by the subtraction some components already averaged from the recorded activity, Vaughan and Ritter (1973) argue that notions of information processing involving serial additive processes are oversimple and that the operation of parallel processors and feedback mechanisms, as well as the physiological state of the subject when stimulated, need to be included. Parallel processing being indicated by components occuring overlapping together at the same electrode (Tueting 1978). What needs to be kept in mind is that a behavioural measure, e.g. reaction time (R.T.), is only an index of an internal process, e.g. attention, as indexed by neuronal activity. This corollary is particularly important in studies involv-

ing endogenous ERP components. The ERP components cannot be regarded as a 'correlate' of some diffuse psychological state, but rather as a manifestation of neural activity which plays a part in informational transactions, and as such are intimately related to psychological processes (Donchin 1977; Donchin et al. 1978).

The usefulness of any psychological process may depend on the level of generality with which it is presented. Thus, Picton et al. (1978) present 'attention' as the process underlying the selection and organisation of available information and intimately linked to general arousal, which controls the availability of both information and response. That different interpretations of attention exist, stressing 'attention', 'alertness', vigilance' is noted by Buser (1980) along with its ambiguous connotation of both the end result of an attentional process as well as the process underlying it. Attention is seen as containing the quantitative aspects, related to the degree of arousal or its activation, and qualitative or selective, which are situation dependent as, e.g. in the orienting reaction. Model building becomes especially difficult due to the variability of subjective engagement in any experimental task. Nonetheless it is generally recognised that subjects can, and do, vary their involvement and this needs to be directly assessed in the experimental situation and contained in any explanatory model of behaviour. Simple instructions to subjects to 'attend' do not suffice (Shagass 1976; Donchin 1977; Rebert 1978; Picton 1980).

Applying such a perspective, Donchin et al. (1978) state that the ERP can be viewed as a sequence of serially linked processes, activated by experimental manipulations, that are manifested at the scalp in distinct positive-negative fluctuations. Analysis of the data is often begun with the identification of the processes or 'components'. Whilst such is normally accomplished by a trained observer, certain inherent dangers in such a proceedure need to be understood. The observer may be biased, his perceptions

distorted by his expectations, and he may tend toward a subjective selection of data in support of personal hypotheses. Besides such dangers, objective data interpretation is recommended by the nature of the recorded signal itself. Positive and negative elements of the ERP are not necessarily independent, and their degree of interaction cannot be gauged by simple inspection. Slower potential drifts, e.g. the CNV as recorded at the scalp, may not be produced by the linear summation of several independent generating mechanisms. As Picton et al. (1978) note, at any one latency, multiple areas of the brain may be simultaneously responding to an incoming signal, and thus be represented by no single component. Finally, to concentrate on only one such feature ignores information contained elsewhere in the ERP.

Factor analysis of digitalised recorded data exists as an alternative to visual inspection of waveforms and offers certain advantages. Data is simplified through being collapsed into a smaller number of meaningful components. As the obtained components are extracted from the data, so experimental bias is eliminated. The components identified may be those responding to experimental manipulation of the variables even though they overlap in time and space. Limitations exist nonetheless. Small, but important, components in the data may be discarded by the parsimony imposed by extracting major components. Bias is not wholly eliminated in that no a priori guidelines exist to direct data selection, the applicable correlation matrix, the type of factor analysis, the number of components to be extracted, or the optimal rotation method. Finally, although factor analysis can extract overlapping factors, it can only do so when their latencies are invariant. Changes in latency will markedly distort factor analysis procedures (Picton and Stuss 1980). This renders it inadvisable to apply, without correction for latency variation, when such variation is to be expected.

Certain comments can be made concerning ERP generators.

Electrophysiological manifestations of the ebb and flow of information transmission between neuronal aggregates appear at a pair of electrodes on the scalp as a spatio-temporal moving dipole, the movements of which trace, between the electrodes, a series of potential changes, the time course of which is represented in the ERP. Although such activity is assumed to represent post-synaptic activity, no assumptions can be made concerning the number, polarity, or orientation of such dipoles (Vaughan and Ritter 1973; Donchin et al. 1978). Nonetheless, the sequence with which such neuronal aggregates are activated and their degree of of interrelation demonstrates the transmission of information or, in other terminology, complex psychological activity. Thus, an ERP component is a subsegment of the ERP whose activity represents a functionally distinct neuronal aggregate. Functionally distinct aggregates need not automatically be anatomically distinct neuronal populations; that is, neuronal groups can serve different functions. Just such a plasticity of neuronal involvement is cogently presented by Luria (1973) particularly regarding the regulation of goal-directed activity. Thus, a component is a set of potential changes that can be shown to be functionally related to one or more experimental variables. Insofar as as it reflects experimental manipulation, it may therefore involve several peaks and troughs, or even the whole waveform.

1.1. The Contingent Negative Variation.

That a large negative shift in the baseline elicited by paired stimuli is still recognised by the name "Contingent Negative Variation", despite the subsequent introduction of various psychological causative and mediative processes, bears testimony to the pioneer work of Walter, which is continued still by his colleagues. This section addresses itself to the early investigations carried out, prior to

1973, on the CNV and the reorientation of research, and modification of its interpretation, subsequent to studies identifying separate components in the waveform. In this section the experimental work prior to 1973 will be introduced and discussed before proceeding to subsequent investigations. Early reviews can be found in Cohen (1969), Tecce(1970, 1971, 1972), and later reviews in Callaway (1975), Donchin et al. (1978), and McCallum (1979). Conferences on ERPs, with the CNV forming a notable part, are reported in McCallum and Knott (1973, 1976), Otto (1978), Lehman and Callaway (1979), and Kornhuber and Deecke (1980). Post-imperative negative variation, not being included as a variable in the present experiment, will not be discussed here.

The occurence of the CNV in the operant conditioning situation with normal subjects "is the most predictable feature of human brain electrogenesis" (Walter 1967). Whilst other researchers have extended the paradigm to other situations, the basic features remain the same. The CNV is a surface negative potential, appearing as a baseline deflection, which occurs between paired stimuli which are physically or internally presented. Early reports (Walter 1966; Cohen 1969) of an onset latency of 200 msec confused late responses to the evoked potential with the CNV, and a latency of around 450 msec is now commonly accepted. Using short interstimulus intervals (ISI), the CNV rises to a maximum at, or shortly after, S2. Under constrained circumstances a further negativity, the post-imperative negative variation (PINV), is found after S2. The CNV is maximally recorded in reaction time paradigms at the vertex (mean: approxiamately 20 μv. S.D.: 4 μv. Range: 5-50 μ v.) to decrease gradually anteriorly and rapidly posteriorly. In other experimental paradigms maximal amplitude may shift to frontal electrodes, but a clear signal is still obtainable at the vertex. It is bilaterally synchronous, but the inclusion of a BP may introduce asymetry in the later part of the CNV. It may be recognised in the raw EEG of some 20%

of normal subjects and in 80-90% of averaged records. In contrast to early and middle EP components, the CNV can be obtained with as few as four trials, 8-12 being the range commonly adopted. Its morphology presents certain characteristic waveforms. Extending the ISI to over 3-4 sec. allows a biphasic waveform to emerge, which otherwise may only be obtained with the application of principle component analysis to the raw data. Variability in CNV latency recommends the calculation of variance. It has been shown that amplitude, latency and variance are not independent, and that variance generally may be of more significance than either latency or amplitude (Tueting 1978). However, the determination of variance at any time point in the waveform does not reflect true variance. Firstly, because it is difficult to separate the variance contributions of time and amplitude. Secondly, variations have a random component as well as systematic changes over time. Finally, as the CNV is extracted from the EEG, it has already obtained a contribution to its variance from this source (Cohen 1974; Tecce 1971, 1972; O'Connor 1980).

The first study on the CNV (Walter et al. 1964) reported a prolonged surface negative component elicited with a motor response to a second stimulus the presentation of which was warned by a first (ISI: 1 sec.), independent of modalities used. Modifying the experimental paradigm by withdrawing the imperative stimulus led to a gradual abolition of the CNV, but its reintroduction permitted a full restoration of negativity. Equivocation in S2 presentation produced a reduction in the CNV. The degree of reduction being a function of the probability of stimulus association; 0.5 probability producing no CNV and, interestingly, a probability of 0.7 being, occassionaly, reported as having the largest associated CNV. In effect the CNV, or 'E' wave (Walter 1966), mirrors the "subjective probability or expectancy of reinforcement" and the "intention to respond in some way to the imperative". Although much of the early work stressed the importance of an operant response, it was

increasingly realised that a mere mental event was sufficient to obtain large and consistent CNVs (Walter 1967, 1975; McCallum 1969) and in the absence of a motor response if, for example, when an experienced experimentee is instructed to estimate a time interval (Walter et al. 1964). The degree to which the subject complies with instructions were also, from the outset, recognised as of central importance. The subject's involvement in the experimental paradigm was early on manipulated by designing intrinsically interesting experiments. The rise in brain potential passing a certain threshold was used to control S2 presentation in the same manner as a motor response; subjects could even monitor their own expectancy wave (Walter 1967). Other studies controlling the content of S2 will be presented later. Recording the CNV with chronic intracerebral and subdural electrodes in patients showed a scalp attenuation of about twofold and it rising in many superficial patches of the frontal cortex (Walter 1967). Localised frontal lobe activity is also described by Luria (1973) in relation to the execution of complex mental tasks.

Whilst modality of the paired stimuli is unimportant, the modality of any distracting stimuli does have an effect. Auditory distraction in the intertrial interval, with a click as S1 delays CNV onset and can lead to a reduction in amplitude. Visual distracting stimuli, which is irrelevent to the current task and delivered in a different modality, has no such effect (Walter 1975). In properly motivated subjects the CNV could be sustained for as long as 20 sec. This phenomenon was regarded as a "true conditioned brain response". Its close correlation with attentiveness and expectancy suggested it to be an electrocortical sign of "cortical priming", whereby responses to associated stimuli are economically accelerated and synchronised to culminate in a final discharge about 120 msec after the imperative stimulus (Walter et al. 1964). McCallum (1969) emphasised the relationship of attention to the CNV, although recognising that the divergence of interpretation was more a

matter of semantics than physiology.

Various descriptions have been advanced to contain differences in CNV morphology. Bostem et al. (1967) identified three typical patterns consisting of a convex dome form, or a square wave with a sharp rise to maximum potential which maintains a steady level, or a progressive rise with a saw-tooth appearance; this last being the most frequent. The second two correspond to the typologies of Types "A" and "B" respectively and presented by Cohen (1969) and Tecce (1970). Type A CNV has been found when subjects are uncertain of the timing of S2 occurence or when S1 contained task relevent information, and Type B in situations of certainty of S2 delivery or when S2 contains information (Tecce 1972; Weinberg et al. 1976a,b). A fourth CNV variant is identified by Dincheva et al. (1971) consisting of a biphasic response. However a full evaluation of this is uncertain as the authors used a 1.5 sec. ISI.

Other psychological processes than attention and expectancy, e.g. conation, arousal, motivation, have been adopted and championed by different research teams in their extention of the operant conditioning paradigm to other experimental situations. Expectancy per se was not seen as the unique psychological correlate of CNV by Irwin et al. (1966a,b). On the basis of conditions comparing no response versus response, low versus painful shock, threshold versus supra- threshold stimulation, and greater rather than less effort in response, they concluded that conditions which increase the energising factors of behaviour, i.e. motivation and drive, also increase CNV magnitude. Motivation was seen to be involved in that the warning stimulus elicited a "conditioned emotional response". However, Straumanis et al. (1969) obtained no difference between low and high shock conditions; the lack of difference may be due to their not establishing pain thresholds individually. Increases in general motivation, induced by higher muscular effort rather than low and by faint rather than moderate

tones, resulted in increases in CNV magnitude (Rebert et al. 1967). McAdam and Seales (1969) demonstated that a monetary incentive increased baseline slow potential. Motivational enhancement through the effect of feedback and control over stimuli, where a MR terminated a series of light flashes rather than a single flash with MR having no effect, is reported as significant (Peters et al. 1970; Milstein and Small 1972). Similarly, significant CNV enhancement was obtained when a fast response could avert a shock (Cant and Bickford 1967). Motivation, as a CNV determinant was dismissed by Waszak and Obrist (1969) who found no differences in CNV amplitude in a competitive situation. Näätänen (1978) argues that the extensive CNV literature supports the concept of the CNV as reflecting mainly motivational factors, however they are labelled. The CNV is seen as reflecting the "conscious effort to increase the level of performance" and other motivational and emotional factors more than they reflect performance processes themselves.

The initial formulation, given by Walter, to the CNV of expectancy has already been noted. Increasing the ammount of information in the S2 promotes heightened negativity in the later part of the waveform (Weinberg et al. 1976a,b). Already noted are experiments in which the subjects monitor their own expectancy wave. Also found to promote larger amplitude CNVs are Raven matrices, monetary incentives, complex visual stimuli, and pictures of naked women as S2 in males. However, on the basis of operant conditioning experiments, Low et al. (1966a,b) concluded that 'conation' was a better descriptor than 'contingent'. Conation was defined as "the conscious drive to perform apparently volitional acts with or without knowledge of the origin of the drive". Using 'conative control' McAdam et al. (1966) were able to produce increases or decreases in CNV by 'thinking' high or low CNV.

Finally, the processes of attentiveness and arousal have been implicated in CNV genesis. Decreasing intensity

of S2 produced increasing CNV amplitude due to the greater degree of attention required (Low et al. 1967). Combining two CNV paradigms overlapping together also produces higher CNVs (Low and McSherry 1968), but not in any simple additive fashion. The reductive effects of equivocation and distraction have already been introduced. Tecce and Scheff (1969) reported that both introducing an irrelevent stimulus into the ISI or giving a second task both serve to decrease CNV amplitude. Changes in arousal or drive were discounted because subjects reported greater effort in distraction conditions during which, in addition, heart rate levels were elevated. Increases and decreases in CNV amplitude to interpolated stimuli containing task-specific information is also reported by Papakostopoulos (1978). Increasing attentiveness to S2 was also associated with elevated CNV (Milstein et al. 1974) in a gambling situation (with tumblers constituting stimuli). Losing was associated with more negativity than winning, and stimuli to which were attached 50% bonuses produced the greatest enhancement of all. Selective attention, involving greater alertness, to faint tones was associated with a significantly higher amplitude for detected rather than missed tones (Hillyard 1969). Anticipation may well also exercise a mediating effect on arousal processes. Thus, the CNV is reduced when subjects initiate the stimulus sequence themselves (Hamilton et al. 1974). McCallum (1976) reported that increasing task complexity first tended to increase CNV amplitude levels but subsequently, with further increases in complexity, CNV amplitude declined. This corresponds with a finding of McAdam (1966) that CNV amplitude increased or decreased according to trials sorted as to early, middle or late responses in a task requiring subjects to estimate a 1.5 sec. interval. Tecce (1972) introduced the concept of the inverted-U arousal hypothesis to account for such results.

Knott and Irwin (1967, 1968) and Low et al. (1971) investigated the hypothesis that high scorers on the Taylor Manifest Anxiety Scale would demonstrate higher CNV amp-

litudes than low scorers, based on the idea that the two groups differed in arousal level. No differences were obtained in conditions in which a MF was, or was not, required or when S2 was a low intensity shock. However, when S2 was a painful shock, high scorers produced smaller CNV amplitude. Tecce (1972) suggests that high anxiety subjects may have been overaroused by the prospect of a noxious stimulus to the detriment of the CNV. In an increasingly difficult discrimination task with performance errors punished, Knott and Peters (1972, 1974) demonstrated that females reduced CNV in amplitude on response trials, and increased it on non-response, as discrimination became more difficult and errors more subject to punishment. Males did not show the same progressive change.

Although multiple peaks in the waveform had been previously reported (Walter 1966; Dincheva 1971), their systematic manipulation as experimental variables is generally accredited to Loveless and Sanford (1973) and Weerts and Lang (1973); both reporting a biphasicity. The preceding preoccupation with short ISIs may be attributable to a reluctance to surrender the classical conditioning paradigm which requires optimally short intervals to obtain the most rapid conditioning (Loveless 1978). To the extent that biphasicity, whatever the psychological processes any components may represent, has now been replicated so often as to be an indisputable electrocortical phenomenon, the problem remains of unequivocally interpreting previous research findings. Insofar as different processes are called in to force by some amalgum of subjective and situational variables, interpretation of early CNV research is made difficult by the precise determination of the effect of such an amalgum on the obtained waveform on any one occassion. Further, no definite statement can be made as to whether, with short ISIs, early and late CNV components would be additively expressed in the waveform, or whether one partly inhibits the expression of the other. Weerts and Lang (1973) suggest that habituation in the early component is

compensated for by increases in the latter so as to produce an apparently stable waveform. The possibility also exists that different electrocortical events are involved as a function of ISI duration. However, inspection of results clearly shows that, with longer ISIs, CNV amplitudes are markedly reduced. McCallum (1979) and Loveless (1978) note that the later CNV component may well be the Bereitschaftspotential.

Already existent at the time of the demonstration of CNV biphasicity were suggestions that the CNV demonstrated a compound waveform. Järvilehto and Fruhstorfer (1970), using a 1 sec. ISI, identified three types of negative potential. A central dominant potential, interpreted as a sign of readiness to act, a frontal dominant occuring in an auditory discrimination task not succeeded by a MR, and a vertex maximal type, preceding a discrimination irrespective of whether this was followed by a MR or not, were identified. The third type was regarded as possibly being the summation of fields from the first two. They postulated that the CNV was composed of the frontal dominant and central dominant components: the former of these, and particularly its duration reflecting uncertainty, is present even in the absence of a MR. In situations in which a discrimination requires a response, these waves coalesce. The ability of subjects, previously trained in the normal CNV paradigm, to generate waveforms of closely similar shape and amplitude to the CNV, in the absence of one or both stimuli and without a MR, suggested that the CNV was not a unitary phenomenon, but could result from the activation of different cerebral functional systems (Loker 1973). Lack of control over internal stimulation by subjects makes evaluation of this experiment difficult. Van Veen et al. (1973) identified two orthogonal components, corresponding to early and late components of the waveform on the basis of factor analysis. It is generally accepted that the second component peaks with S2 presentation, and the first at 750 msec (Gaillard and Perdok, 1977) or one second (Loveless 1976) and decays

over some 5 secs. (Loveless 1978).

Applying d.c. recording to an 8 sec. ISI, Weerts and Lang (1973) obtained a biphasic waveform in the CNV. An early negative component (-12 μv), which habituated over trials to reach -8 μv, was interpreted as a cortical component of the orienting response to the signal stimulus. The later negative wave was assumed to be the classical CNV and increased in size over the experimental session. When corrected for eye-movement, however, these changes were nonsignificant. Loveless and Sanford (1973, 1976) using ISIs ranging from 0.5 to 15 sec., distinguished two components in the CNV irrespective of duration; the first was equated with the orienting response (O wave) to S1 and the second (E wave) with anticipation of S2. Older subjects did not show this anticipatory negativity with longer ISIs, but rather produced a CNV of sustained low level throughout the foreperiod. This suggested that they used the warning stimulus merely to increase their general level of alertness (Loveless and Sanford 1974). The orienting and expectancy interpretation of the biphasic form is supported elsewhere (Peters 1977; Peters et al. 1977, 1978; Lang et al. 1978; Loveless 1977). The orienting reaction is a behavioural response to stimuli which force a response (Sokolov 1963), an important function of which is to facilitate responsiveness to subsequent stimuli (Loveless 1977). The identification of the O wave with the orienting reaction was based on their topographical similarity and mutual dependence on a warning stimulus; which is reviewed below.

Assuming that the components reflect different processes, then they should respond selectively to experimental manipulation. Loveless and Sanford (1975) demonstrated that varying S1 intensity and ISI duration both significantly increased peak amplitude of the O wave, but neither affected the E wave. The effect of ISI duration they accounted for as due to a systematic distortion in their one second ISI condition in which the two waves combined. Significant increases in the O wave amplitude to S1 duration and

intensity, and in both to ISI duration, were interpreted as supporting this result (Klorman and Benson 1975), but they were unable to demonstrate changes in components over time. Holding the warning stimulus constant, Loveless (1977) observed no differences in O or E waves in a comparison of simple against choice reaction time. The lack of difference between key and non-key stimuli was ascribed to a conditioned defensive reaction to the intensive auditory non-key stimulus. That no difference could be observed between simple and choice reaction time situations was seen to mitigate against the interpretation of the E wave in terms of expectancy in that this wave was unaffected by uncertainty. Gaillard (1976) demonstrated that auditory over visual stimulation had a significant affect on the O wave, while the late component was reduced by lengthening the ISI; the amplificatory effect of auditory stimulation being confirmed elsewhere (Rohrbaugh et al. 1979). Within trials, amplitude of the O wave remained constant while the E wave decreased in amplitude. These waves were regarded as orienting and motor preparation responses respectively. The orienting interpretation of the O wave is further supported by its reduction to noise distraction (Loveless 1978). Auditory S2 presentation produces increased E wave negativity at the vertex in a reaction time paradigm (Gaillard and Näätänen 1976). The same interpretation was made following an experiment in which instructions emphasising speed of response, but not probability of S2 presentation, led to increased late component negativity (Gaillard 1977). Significant correlations between S2 probability and CNV amplitude and R.T. were earlier reported by Jarvilehto and Mantysalo (1976). The necessity of motor preparation was posited for late negativity development where no such component was present in a detection paradigm (Gaillard and Perdok 1977, 1979). In the latter report amplitude of the early wave was increased when S1 contained information relating to the discrimination task. Terminal CNV was not related to peceptual efficiency.

An overt motor requirement is elsewhere reported as reliably associated with the presence of a distinct anticipatory component (Simons et al. 1979; Fröhlich et al. 1980). This latter study reported no CNV in a "no-go" situation, but it is suspect on several grounds: namely, recording only from parietal locations, no monitor of eye movements and instructing subjects to keep their eyes closed. Peters et al. (1978) reported that the two components of the CNV are differently capable of predicting R.T. at different points in the series. Electrocortical manifestations of motor preparation were comparatively examined in a study including the CNV by Grünewald et al. (1979), who concluded that the BP exhibited the same influence of speed of response instructions as the late component of the CNV. Amplitude patterns at the scalp demonstrated positive intercorrelations indicating comparable influence of functional subjective factors. These authors were able to confirm a more anterior CNV distribution only for the early component. They did confirm, however, the lateralisation effect of the BP, and supported McCallum's (1979) record of BP amplitude at around -5 μv. Both the CNV and BP are reported to be responsive to increases in motivation (Waszak and Obrist 1969; McAdam and Seales 1969; McAdam et al. 1971). Using a 6 sec ISI, Larbig et al.(1982) demonstrated that negativity of the late component, but not the early, increased across conditions which introduced an aversive and unavoidable S2 which subjects attempted to contain with any pain coping strategy that they knew. EM and muscle artifact were discounted as contaminants of the record. The authors concluded that "the late negative shift is sensitive to motivational relevance.....of anticipated stimulation but depends also on a tonic component.....determined by the anticipated future presentation of shocks".

Investigating unpaired visual and auditory stimuli, Rohrbaugh et al. (1979) observed that the subsequent negative after-wave and the early CNV wave had essentially

identical appearance and topography, but came to no conclusions as to whether this formed part of the evoked potential or a non-specific cortical function reflecting transitory alerting, orienting, arousal or activation. In an earlier study (Rohrbaugh et al. 1976), they reported an early CNV component, associated with the warning stimulus, and a late wave, related to reaction time which demonstrated higher negativity contralaterally to the responding hand. This latter component they identified with the readiness potential. Whilst the caution of Loveless (1978) that all muscular potentials should be controlled is noted, nevertheless pre-S2 negativity is unlikely to reflect solely motor preparation (Donchin et al. 1972, 1974, 1978). The later CNV component is taken by Lang et al. (1978) as the true CNV. Using "high interest slides" of attractive naked females, over "low interest" of household items, they obtained significantly higher late negativity which, although reduced by the removal of the MR requirement, remained of significantly higher amplitudes. Since the occurence of a motor response is regarded as of some significance, mention can be made here of studies reporting on the nature of the response. Otto and Leifer (1973) found that negativity was more reduced with a double, rather than a single, response task to S2, i.e. one involving a button depressed during the ISI. This was confirmed by McCallum and Papakostopoulos (1972), but not when the button was pressed well before S1.

Two components in the CNV have been reported (McCarthy and Donchin 1978; Donchin et al. 1977) in an experiment (1 sec. ISI) comparing structural and functional matching; the independence of these being confirmed by principle component analysis. That there are two distinct components, one motor and the other representing cognitive preparation (Donchin 1975), is supported by Poon et al. (1974) who identified a parietal dominant CNV with problem solving activity and a central dominant in a disjunctive reaction time task.

Changes in CNV topography, in a R.T. task during which

S2 probability was controlled, indicated that at least two different CNV components existed (Järvilehto and Mantysalo 1976).[1] Gaillard (1976) accounts for the Type A CNV (Cohen 1969) as containing a prominent O wave, which is absent in the Type B. The early component is normally recorded maximally at frontal derivations and minimally at parietal (Klorman and Benson 1975; Rohrbaugh et al. 1976; Gaillard 1977). The late component is recorded maximally further posteriorly; precentrally by Rohrbaugh et al. (1976) and non-significantly more in parietal leads (Klorman and Bentson 1975). The CNV is posited as containing three components by two groups of researchers. The first reflecting orienting or the inhibition of a response, the second as formed by the cortical involvement necessary for the motor or perceptual response, and the third, a generalised motor readiness or organisation of behaviour (Picton et al. 1978; Simons et al. 1979; Picton 1980). Introducing a cautionary note, Donchin et al. (1978), accepting that such studies underline the differential involvement of cortical areas involved in preparatory and associative processes, state that "the attempt to replace one diffuse and generalised effect with two localised and slightly more specific effects would seem to be an exercise of dubious merit".

The foregoing paragraphs are by no means exhaustive of the experimental results obtained. Precisely which psychological construct is applied is a matter of arbitrary choice in that, with proper conceptualisation, any one may be made to fit. As Knott (1972) observes, probably no one correlate exists and slow potential behaviours should be studied in relation to interacting psychological variables. However, the concepts that researchers have attempted to apply to electrophysiologically obtained data are themselves nebulous. Cooper et al. (1978) argue that attempts to link CNV changes to existing psychological constructs have applied such terms as attention, motivation, expectancy, subjective probability which, demonstrating a significant overlap in definition and usage, all lead to a well-recognised but

ill-defined area of human cognitive function. To encompass all such terms, as well as the subjective awareness of them, they present the term 'scopeutic'. This describes a selective state of continuous involvement with a particular set of circumstances which has taken over the central processor for a given period of time and which require at their end-point some action or decision. Physiologically, the scopeutic mode of processing is reflected in slow waves. However, to account for the absence of slow wave activity in situations in which the subject is deemed to be acting automatically, the term 'categoric' is also posited. Such a pre-set pattern of responding is seen to be the result of previously repeated occurences in the scopeutic mode. Whilst this latter mode is flexible in its adjustments, it is likely to be overloaded; features which are reversed in the categoric mode. The scopeutic mode can monitor, or be reapplied to, situations in which the categoric mode is operating. Such may explain the acquisition, gradual decline in amplitude and later recovery, particularly with the introduction of novelty, of CNV in some experimental studies. That involvement is continuous allows goal-directed activity to be modified. Thus, Papakostopoulos (1978) and Papakostopoulos and Jones (1980) demonstrated that introducing a 'Don't press' signal between S1 and S2 led to the abolition of CNV. Clearly transition from one mode to another would depend on the individual and on situational demands. Transition is not automatic and, hence, Näätänen and Gaillard's (1974) inability to demonstrate habituation is not out of place. The reapplication of scopeutic processing is, presumably, equivalent to the "rubbish stores" (Walter, 1975) which serve as a repository for past events against which current are matched for similarity or change.

Other models of CNV acquisition and environmental responsiveness have also been proposed. Walter (1973) posited that slow potential changes presented an outward sign of electrochemical processes underlying the capacity of the non-specific frontal cortex to integrate information

from sensory sources with internal stores, and as such reflected short-term memory involvement in the forming of decisions and planning of action. However, whilst anticipation of further stimuli is undoubtably involved in CNV production, it fails to fully account for the variability of the phenomenon in different circumstances. Its magnitude is changed by factors that leave unaltered the statistical or contingent probability of S2 presentation. Such factors could, for example, index the subjective importance of involvement. Accounts of its genesis stressing motivation are too specific and so fail to account, for example, for its reduction in distraction conditions when subjects report increased effort. Perhaps what fails in these accounts is any recognition of responsiveness to a general setting in which specific tasks are presented. Tecce (1970) suggests that it may be profitable to replace the concept 'motivation' with that of 'attention'.

A two-process explanatory model incorporating attention and arousal was proposed by Tecce (1971, 1972); this being commonly referred to now as the 'distraction-arousal hypothesis' (Tecce 1976). Attention is considered a hypothetical organismic process, characterised by steering functions, which facilitates the selection of relevent stimuli from the environment (both internal and external) to the exclusion of other stimuli. CNV studies show that its magnitude bears a clear monotonic relationship to the experimental task. This association is highlighted by studies involving both phasic and sustained distraction, and it is most clear-cut when there is objective evidence that both the distracting stimuli can be recalled and that the processing of S2 is impaired, i.e. when R.T. is delayed (Tecce 1979; Tecce et al. 1976). Other stimulus variables found to encourage a marked CNV development include a barely audible stimulus as S2 (Rebert et al. 1967), and the use of novel, varied or attractive stimuli (Milstein et al. 1974). Further, defective CNV development and stability is observed in groups recognised for their inability to sustain attention, e.g.

schizophrenics, children, high anxiety neurotics. The inclusion of arousal processes is necessitated on two grounds. First, recording with chronic implanted electrodes in animals and man involve the reticular formation and thalamus in CNV genesis. Second, the liklihood of a time related increase in phasic arousal in anticipation of S2 may affect CNV development. Arousal is here considered devoid of any steering functions, but rather energises behaviour unselectively, affecting only the intensity of the response. CNV magnitude may be selectively affected by such processes as attention or motivation in addition to the arousal-derived contribution. Näätänen (1978), however, observes that correlations between CNV measures and performance have generally been rather low or even nonexistant. Whilst favouring such explanatory models as involving motivation and emotion, he notes that above a certain level, such factors have no consistent relationship with performance.

Tecce (1972) also presents an alternative model, emphasising arousal, to account for the variety of experimental results. In this, the CNV is argued to have a non-montonic relationship to arousal level; a neurophysiological application of the inverted-U hypothesis of arousal. Supportative evidence is found in results showing increased CNV, with increased attentiveness to S2, and reduced CNV with phasic and sustained distraction, at the same time as increased tonic arousal as shown by overall heart rate level (Tecce and Scheff 1969). Similarly, increased CNV amplitude was associated with decreased phasic arousal, as demonstrated by decreased heart rate variability (Lacey and Lacey 1970). Noting that activation, manifested in part in behaviour, combines specific (directional) and nonspecific (general) aspects, Näätänen (1973) states that claims for an inverted-U relationship between different values of different activation measures, or their combination, with performance rests on inadaquate experimental evidence. Explicit in the inverted-U hypothesis is the premise that an optimal level of activation allows an optimal level of

performance. He argues that the subsequent degradation of performance with increasing activation is not dependent on activation per se, but rather due to changes in direction, or the qualitative aspects, of activation away from the task at hand. Divergent demands are thus made on the subject by the presence of other tasks, stimuli, possible reactions, or by other organismic states than the basic.

McCarty and Donchin (1978) present a metaphorical "Terrain" hypothesis to account for the dissociation between CNV measures and performance. Any such association depends on the physiological and psychological terrain which the organism is encountering. According to the hypothesis, CNV variance represents adjustments, in the preparatory systems, that are made by the organism to acheive a fixed behavioural output in the face of varying conditions. Exactly in the same way as the car's accelerator pedal is regulated to maintain a steady speed over differing road conditions, so, it is hypothesised, that the processes manifested by the CNV might constitute a manifestation of a control system to keep constant some behavioural output. Variance is due to changes in organismic processes (Donchin et al. 1978). No direct evidence in support of this model is advanced.

Noting the dissociation between R.T. and CNV measures, Loveless (1973, 1978) observes that preparation for S2, which develops consequent on S1, ensures that these responses fall into a narrow band, except when subjects anticipate or fail to respond. The slow potential following S1 serves an executive function of differentiating signals from background activity and, on the basis of comparisons with memory, identifying warning stimuli, i.e. those requiring action. Such serve also to increases the probability of detecting subsequent stimuli. High correlations cannot be obtained due to the restricted range

1.1.1. Sources of artifact in the CNV.

Various possible sources of artifact have been identified and these need to be eliminated through methodological procedures in order to obtain valid results. Perhaps the most serious threat comes from eye-movements (EM) (Hillyard and Galambos 1970); this is particularly the case with psychiatric patients (Straumanis et al. 1969). Indeed some researchers (Tecce et al. 1976) include these in their behavioural evaluation. That the CNV is an authentic electrocortical phenomenon, though, is supported by recordings with intra-cerebral or subdural electrodes (Walter, 1967; McCallum et al. 1973, 1976) and with patients with no eyes (Low et al. 1966b). In addition, electrodermal, heart rate and respiratory influences will be reviewed. Roth (1977) observes that the same sources of artifact that affect the CNV pose a potential threat to the P300 also; although discounting skin potential changes and CNV resolution as generators for the P300 (Roth 1978).

Within the eyeball is a standing potential of several millivolts - the corneo-retinal dipole - oriented with the cornea positive to the posterior portion of the eye. The electrical fields generated may be recorded over the entire scalp to the extent that eye rotations can endanger potential shifts at the vertex by producing larger potentials than that of the vertex. Downward eye-movement, producing a negative potential, is particularly hazardous because, when synchronised with experimental stimuli, produces a waveform indistinguishable from the CNV: indeed, 'pseudo-CNVs' can be generated in the absence of any stimuli. The shift produced is largest at frontal electrodes and diminishes posteriorly. Analysis suggests that EM artifact is added to the CNV in proportion to its shift (Straumanis et al. 1969). Hill (1974) suggests that the quantitative amount of this contamination, induced in the CNV from this source, can be estimated by constructing a "calibration" function that relates the size of electro-oculographic (EOG) deflection to

the concurrent artifact at the vertex. Algebraically subtracting upward from downward (positive from negative) EM eliminates the CNV and yields a remainder of twice the potential for that shift. An ocular potential of 125 μv. was calculated to produce an artifact at the vertex of 23.8 μv. (some 20%). A similar result was obtained for induced artifact from EM with eyes closed.

Whilst eye-movement produces one sort of artifact, eye-blink offers another. Holding the eyes open in any one position may change the corneo-retinal field configeration. Blinking, squinting, and pulling the lid manually across the eye, all produce large d.c. potential shifts in the vertex EEG with field spread into the vertex-mastoid channel. However the ratio of squint-induced artifact is much smaller than during voluntary movement. The location of the response-key, rather than the location of stimuli, are reported to determine vertical and lateral eye-movements (Weerts and Lang, 1973).

Various techniques exist to eliminate EOG artifact. Artifact calibrations, as noted above, are impractical to establish for each subject due to time constraints. Further, eye-movement induced artifact may not have the same relationship to vertex-artifact as lid-movement, squint, or eye-movement having a different lid closure. It is reported that artifact can be eliminated by the procedure of connecting one pole of a potentiometer to an electrode above the eyes and the other to the mastoid (McCallum and Walter 1968). The central gauge can then be set to zero to balance EM artifact at the vertex. This method suffers certain disadvantages. Movement-related potentials may not have the same artifact-to-EOG ratios so that correcting for one may not eliminate artifact from the other source. Also, no gaurantee exists for a strictly linear relationship between the EOG shift above the eyes and the artifact at the vertex for all kinds and sizes of EOG deflections (Hillyard 1974). Weerts and Lang (1974) found that eye-blink potentials diminished twice as much as eye-movement at the vertex.

Further, since cerebral potentials can reach electrodes for the EOG, they may cancel out when potentials derived from the EOG are subtracted from the vertex (Dubrovsky and Donchin 1978). Since lateral eye-movements may also be involved, the situation is even more complicated. Another alternative is presented by recording from sites insensitive to EOG artifact, e.g. a temporal (Hillyard 1974) or parietal (Weerts and Lang 1974) site. Both recognise that a large degree of signal attenuation results; possibly up to 60% (Hillyard 1974). The possibility of merely attempting to equate experimental sessions in terms of EOG artifact is vitiated by the variability of the EOG, by the difficulty of identifying the degree of contamination in any averaged record, and by the fact that only relative, rather than absolute, measures are so derived (Weerts and Lang 1973; Hillyard 1974). Finally, in order to obtain absolute measures, visual fixation is recommended with the discarding of damaged records. Most adults are able to fixate their eyes for relatively long ISIs, particularly if blinking is permitted in the ITI. Exclusion, either by on-line computer analysis, or visual inspection, is acheived according to some pre-determined criterion. Lateral eye-movement are not seen as so great a threat to the vertex-derived EEG, and can in the same fashion be contained. Whilst this method seems to be the least susceptible to systematic error (Hillyard 1974), it is expensive in terms of discarded data. Such a process may systematically exclude particular groups of individuals (Roth 1977). CNV amplitude may be diminished insofar as a further task requirements is introduced to subjects (Weerts and Lang 1973).

Galvanic skin potentials (GSR) on the scalp may approach the order of millivolts and, although their latency and time course are different to that of the CNV, conditions may arise when conditioned skin reflexes infiltrate the CNV (Hillyard 1974). Latency of the CNV is around 450 msec and that of the GSR about 1.5 sec. with a rise time of about 1 sec. CNV can be seen to which GSR changes do not contribute

and, whilst the former may decline in certain paradigms, GSR typically declines over the course of the experiment (Low et al. 1966b). Greenfield et al. (1963) reported a decreased responsivity in physiological measures, including GSR, in those scoring highly on the Depression Scale of the MMPI. Skin conductance and potential were found to be independent of heart rate changes and unrelated to the CNV (McCallum and Papkostopoulos 1973). This is in accord with Dincheva et al. (1971). McCallum (1979) concluded that, with an ISI of 1.0 to 1.5 sec., GSR poses no contamination threat, but may do so with longer ISIs and in studies designed to investigate the PINV. Giedke et al. (1980), in a CNV study (ISI: 2 sec.) of depressed patients and controls, measured GSR in the period 0.5 to 7 sec. post-S1. Only in the response condition, and not in the rest or no-response conditions, did GSR discriminate between the two groups; with controls significantly increasing the number of their responses. However, they reported significant correlations for patients in the response condition from frontal electrodes between CNV and GSR amplitudes. No significant associations were found for the vertex, nor with the PINV. Dissociation of the CNV and GSR is in agreement with Timsit-Berthier et al. (1976). Simons et al. (1979), using a 6 sec. ISI, reported that only in the response condition to high interest S2 (pictures of naked females) and not to low interest S2, nor in the absence of a MR, were skin conductance responses enhanced, and that even this effect disappeared in later trial blocks. The cortical and skin conductance measures were seen to reflect experimental variables uniquely; each being sensitive to different aspects of information processing required in the individual experiments. It is worth noting that scalp GSR cannot be recorded simultaneously to the CNV.

Although heart rate (HR), on the basis of latency and time course, has been ruled out as an origin for the CNV (McCallum and Walter 1968), certain interesting observations can be made. Connor and Lang (1969) reported CNV amplitude

was related to HR acceleration and deceleration as measured in the S1-S2 interval. A tonic acceleration of HR, superimposed on which being a transient deceleration, is reported in an experiment involving increasing degrees of task complexity (McCallum and Papakostopoulos 1973) and by Lacey and Lacey (1970). A tri-phasic response is reported (Simons et al. 1979) consisting of an initial deceleration, acceleration, and secondary deceleration in two experimental conditions using a 6 sec. ISI, but was found to be independent of CNV measures. This corroborating earlier reports of tri-phasicity (Weerts and Lang 1973; Lang et al. 1978) which was unrelated to loudspeaker location or to the presence or absence of fixation lights. Agreeing with Lacey and Lacey (1974), the latter group suggest that the data indicated an association between heart rate and a motor response, but that cortical and cardiac responses may either vary in parallel or be completely independent.

Whilst the vertex area itself is completely free from underlying muscles, electrodes at this site can record myogenic activity of reduced amplitude from active muscle sites. Such activity has been traced to innervation of the superficial subcutaneous layer of muscle in the scalp, to deeper cranial muscles, and to the neck muscles which react to high intensity sound (Picton et al. 1974). However, such has not been shown to be a source of systematic bias in the CNV. Whilst fast activity, without damage to the averaged record, can be removed by filtering, body movement can be controlled by ensuring a comfortable recording position for subjects, by instructions, and by the rejection of any contaminated records. Changes in respiration have been found to be unrelated to the CNV (McCallum and Papakostopoulos 1973; Low et al. 1966a).

Finally, the determination of baseline needs to be considered. This d.c. offset potential may ammount to many millivolts and represent a complex summation of potentials developed across the recording interfaces and innumerable biological membranes. There is no firm consensus as to

whether this potential remains stable for each individual, even during arousing stimulation, or changes in response to a host of psychological variables (Hillyard 1974). One source of artifactual disruption of baseline is from inadaquately chlorided or insecurely applied electrodes. Weinberg (1975) suggests that this standing potential may be higher with depressives. Quantification of evoked potentials requires that the baseline amplitude be known and stable. The only way in which it may be known is if it is set at zero by averaging. Of theoretical interest only, determination of individual baseline require averaging the electrocortical activity throughout the subject's life. Practically researchers refer evoked potential activity in the averaged record to the pre-stimulus baseline of the trials averaged. An alternative being to average activity in all non-stimulus epochs. Verhey (1980), in a study of psychiatric patients, reported that taking a 0.5 or 1.0 sec. pre-S1 baseline did not alter the pre-S2 CNV mean or standard deviation. Measuring activity both before S1 and S2, Gaillard and Naatanen (1973) reported that 'CNV' before S1 could be observed only with short ISIs of 200 or 400 msec In conclusion, it may be stated that a valid CNV signal may be obtained, that is artifact free from external generators, when adaquate safeguards are taken to contain or eliminate those from ocular or myogenic sources particularly.

1.1.2. Pharmaceutical effects on the CNV.

This review is stimulated by the postulated involvement of neurotransmitters in the generation of the CNV, on the one hand, and, on the other, interest generated by the monoamine theory of affective disorders, which is reviewed in a later section. For the sake of clarity, drugs will be presented within their pharmaceutical class. However, although few investigations have been conducted to date,

results call into question studies with psychiatric groups claiming no difference between those receiving medication and others. Clearly a drug-free baseline is necessary before assertions of minimal drug effect can be made.

Abstention from coffee and tea for 36 hours was observed, in two subjects, to lead to CNV amplitude reduction, which was restored to normal levels following ingestion of caffeine citrate (Walter et al. 1964). Caffeine is regarded as stimulating all parts of the cortex. Ashton et al. (1974) demonstrated that caffeine (300 mgs.) produced significant rise in CNV area about 20 mins. after ingestion, but they lacked a control group. In a controlled experiment, Jannssen et al. (1978) administering 150 and 300 mgs. of caffeine citrate, found a significant decrease between pre-drug and the first, of a series of four, post-drug measures. White noise produced a decrement in CNV magnitude only for introverts and not for extraverts. They concluded that CNV effects are independent of dosage.

The effects of nicotine involves the midbrain reticular and limbic systems. Human EEG studies have usually indicated a stimulant effect, despite subjective reports of both alerting and relaxing effects. Cigarette smoking has been associated with both increased and decreased CNV area. This effect is in part dose, and in part personality, dependent. The eight more extraverted subjects in one study apparently had a lower rate of nicotine uptake which facilitated CNV development; the reverse being true for introverted subjects. That is, a higher nicotine uptake per unit of time was associated with a decrease in CNV. Sham smoking an unlit cigarette produced no CNV change (Ashton et al. 1974). Smokers were concluded to unconsciously select different doses of nicotine from the cigarette so as to obtain some optimal dose that met their own personal requirements which were determined by circumstances and personality. A further study, using intravenous nicotine injections (Ashton et al. 1978) replicated these results. Administration of a single dose, produced an increase in CNV magnitude in some

subjects, dependent on their smoking habits, but a decrease in others. Increasing dosage produced an increase in CNV initially with increasing dose, but further increases in dosage led to increasingly larger reductions in CNV magnitude. At the post-drug measure of a saline control, CNV magnitude returned to normal.

Similar quixotic effects are noted with amphetamine, which is considered to have an activating effect on the brainstem reticualar formation. Although CNV amplitude was depressed after 30-50 mins., its magnitude in subjects with slow CNV rise times increased 1-2 hours after injection and was associated with curtailment of eye-blink and subjective reports of easier acheivement of task requirements (Walter et al. 1964). The depressant effect of this drug is reported also by Tecce and Cole (1973), who concluded that the CNV is a "sensitive and accurate indicator of changes in psychological functioning produced by amphetamine".

By contrast, the depressant 'nitrazepam' (Mogadon) has a depressant efect on the brainstem reticular formation and limbic systems. A dose of 2.5 mgs. was found to reduce CNV magnitude after 20 mins. and maximally after 35 (Ashton et al. 1974, 1978). Similar results are reported by Jannssen et al. (1978) for the depressant 'chlordiazepoxide' (10 or 20 mgs. dose). Small CNV reductions under the influence of the hypnotic 'Dalmane' are reported (Hablitz and Borda 1973).

The effects of atropine, metochlopramide and thymoxamine, which are neurotransmitter antagonists of acetylcholine, dopamine and noradrenaline respectively, are reported (Thompson et al. 1976, 1978). Although using small sample sizes, atropine was found to depress CNV amplitude reaching a maximum at 16-22 mins. post-drug and with partial but irregular recovery thereafter. Similarly, the depressant effect of metochlopramide was maximal after 36 mins. Neither thymoxamine nor placebo produced any effect. The authors conclude that both cholinergic and dopaminergic systems are involved in CNV generation.

1.1.3. Reliability of the CNV.

Tecce (1970) suggested that attention, as reflected in the CNV, may be considered a trait variable and, as such, it may be applied to the measurement of individual differences. Support for any such contention must be found in intra-individual stability measures. It has been asserted that the CNV pattern is maintained as long as the subject is attentive and responds promptly (Walter et al. 1964; Walter 1966), and that the CNV remains stable over months (Walter 1965). Again, on apparently visual analysis, Cohen (1969) reports in a single session of several hundred trials and lasting three hours, there appeared no systematic change in CNV amplitude. Systematic investigations have been made of both the intra-, and inter-, session changes, which will be reviewed separately.

A study, often cited as indicating instability in the CNV, by Straumanis et al. (1969) found poor test-retest reliability (TRR) with normals and even poorer with a heterogeneous psychiatric population. What is overlooked is that subjects were assessed in different experimental conditions, albeit in the same experimental session, and that, as the authors note, eye movement presented a serious source of artifact particularly with the patient group. Before any conclusions can be offered, that CNV responsiveness is not a relatively stable individual characteristic, identical experimental paradigms must be applied. Roth et al. (1975), at the same time as controlling for eye-movement, examined the responsiveness of subjects in recordings 30-60 mins. apart and at weekly intervals. CNV amplitude was measured 600, 800 and 1000 msec post-S1, and yielded overall correlations of 0.72 for the same day's recording. The measures were obtained from curves smoothed, by two experienced raters, between a point one second prior to S1 to S2. Any possibility of non-systematic bias is excluded by the high

(r=0.96) correlation between their quantifications. Tecce (1979) found no significant differences in CNV amplitude between two control paradigms, which were separated by a short-term memory task, using the same S1 and S2 stimuli throughout (ISI:1.5 sec.) and lasting 7 mins.: rest periods of 7 mins. each being taken between runs. Within recording sessions, normal subjects reliably maintained their CNV morphology, predominantly showing immediate post-S2 return to baseline (Timsit-Berthier 1976). In a further study (Timsit-Berthier et al. 1980), the effects of long-lasting (1 hour) recording sessions were assessed with weekly intervals. A one sec. pre-S1 baseline was compared with negativity 500-700 msec and 1300-1500 msec post-S1 (ISI:1.5 sec.). A simple MR to S2 was required throughout. Six successive runs of 12 trials each were compared in each recording session. An apparent post-S1 amplitude decrease was not significant, but the pre-S2 reduction did reach significance when comparing runs within recording sessions. Four of the nine subjects recorded showed no change in CNV amplitude in session one. In the second and third sessions, however, all subjects showed pre-S2 reductions. Using a longer ISI, Weerts and Lang (1973) report non-significant changes in early and late CNV components within recording sessions when corrections were made for eye movements.

Turning to reliability over longer periods of time, Cohen (1969) reports a retest of 34 subjects, using a short ISI, producing a correlation of 0.80. Abraham et al.(1976), in a comparative study with psychiatric patients, reported that mean acquisition amplitude for normals was consistent upon retesting but that distraction amplitude increased at the same time as intersubject variability decreased. However, inspection of their tables shows that, for normals on retesting, acquisition amplitude decreased from -18 μv to -2.5 μv. Correlational results on the measures used is not reported. Roth et al. (1975) found test-retest reliabilities over weekly intervals was 0.68 with an inter-rater reliability of 0.98 (see above). No long term changes in

post-S1 negativity was observed by Timsit-Berthier et al. (1980), but pre-S2 negativity decreased significantly across weekly recording sessions. Roth et al. (1975) conclude that CNV reliability is higher than a purely state variable, but lower than that desirable for a trait variable.

These studies are problematic to interpret. Insofar as the CNV is regarded as composed of separate processes or components, any study of reliability needs to study them separately. Whilst Timsit-Berthier et al. (1980) measure early and late CNV amplitude, their short ISI makes it likely that the effects of contributory processes are confounded. Lack of finite conclusions may be partly brought about by recording at different times of day. Thus, cyclic variations in amplitude have been reported suggesting a basic underlying rhythmic process (Borda and Hablitz 1974). This suggestion is supported by a study of Kerkhof (1980) reporting a circadian rhythm in the CNV with a trend towards highest negativity at 10 am. Avoiding paradigms designed to facilitate habituation (Timsit-Berthier et al. 1980) and standardising recording for time of day suggest themselves as necessary conditions for optimally investigating CNV reliability.

1.2. On the relationship between the CNV and P300.

This discussion is introduced here to act as a bridge to a fuller presentation of relevent P300 literature. "P300" is taken to denote the major positive component following signal presentation: the majority of studies being concerned with positivity following S2 in a CNV paradigm. The terms P300, P3, late positive component (LPC), and late positive complex (LPX), adopting the convention used by the author, are taken here as synonymous. However, before considering the extent to which the two are wholly independent, reflecting distinct psychological processes, or related and reflecting, however mediated, the same process,

there is some suggestion that the CNV and P300 are in fact composites of the same phenomenon, i.e. P300 depends on CNV resolution or is modulated by it (Wilkinson and Ashby 1974; Wilkinson and Seales 1978).

That the P300 reflects only CNV resolution was first suggested by Donchin and Cohen (1967). They observed elevated P300 and CNV-like waves, before the stimulus, to single stimuli stressed as important by instructions. Their interpretation of a common attentional involvement was supported by Hillyard (1969) in an experiment demonstrating increased CNV and P300 amplitudes to detected, in comparison to missed, tones. Confirming the results of Näätänen (1967, 1975), Wilkinson and Lee (1972) and Wilkinson (1976) reported that differences in CNV amplitude between relevent and irrelevent stimuli correlated with corresponding differences in amplitude measured 300 msec post-S2. However, their use of a short inter-trial interval may not have allowed sufficient time for the resting baseline to be re-established. Further discussion of P300 as CNV resolution can be found in Karlin (1970). In a further paper (Wilkinson and Ashby 1974), they reported an experiment in which task relevent and irrelevent tones were presented alternatively or unpredictably at one sec. intervals. On the basis of their finding of higher correlation between P300 and CNV for relevent (r=0.78) over irrelevent (r=0.35) stimuli, the authors concluded that the relationship depends not only on the stimulus baseline level but also on the degree of stimulus relevence. The P300 was interpreted as constituting a reactive return of the CNV to baseline. However Wilkinson and Seales (1978) modified this position by suggesting that P300 may be modulated by summation with the positive-going resolution of CNV negativity. Defining P300 as a function of prior state and subjective reactive change to stimulation, they drew support from a vigilence experiment which showed, firstly, that CNV amplitude decreased significantly over trials and correlated positively with P300 level, and secondly, that time on task correlated

negatively, but response confidence positively, with P300. Comparing P300 and CNV in different conditions of stimulus evaluation, Wilkinson (1980) reported that the 'hit' minus 'correct rejection' differences were greatest at parietal electrodes while the CNV was vertex maximal, and that the P300 to 'hits' was larger at all electrode sites than 'correct rejections'. He concluded that the hypothesis that the effect of response category on the P300 was due solely to differential CNV resolution could not be sustained. Further, whilst both P300 and CNV decreased over trials, differences appeared the same at Pz and Cz, but confidence limits allowed no definite conclusions: the CNV resolution hypothesis for the P300 predicts greater differences at Cz.

Preparatory state was controlled in an experiment by Desmedt and Debecker (1979a) by using a variable ISI (mean: 2.5 sec. Range: 1-15 sec.) and randomly presenting target stimuli, either binaural clicks or somatosensory stimulation to one index finger, allowing correct detection on 90-95% of trials; one stimulus type being the target on a particular run. A 'P350' was elicited only to the stimulus designated as target and presented documentation of "a clear situation in which P350 cannot reflect pre-stimulus events and must be related to post-stimulus processing". In a further experiment (Desmedt and Debecker 1979b) a fixed ISI of 2.5 sec. and equiprobable presentation of target stimuli -as before, either a click or somatosensory stimulation- was used. A slow negative potential shift was observed, both to target and non-target stimuli, and the P350 only to targets. On the basis of a rough estimate of the slow potential shift of 0-3 μv, the authors obtained a positive correlation with P350 amplitude. In both reports, contamination from ocular and muscular sources were discounted. In combination, these two reports demonstrate that either the SPS, as a variant of the CNV, or P350 (P300) can occur without the other and that P350 amplitude cannot be accounted for solely in terms of CNV resolution even when both are coterminously in evidence.

Further disconfirming evidence comes from Hillyard et

al. (1976) who identified CNV and P300 as different phenomena on the basis of topographical analysis. Various researchers have reported that P300 amplitude can vary independently of the presence or absence of CNV (Donchin et al. 1976a) or of fluctuations in CNV (Karlin and Martz 1973; Marsh et al. 1976; Poon et al. 1974). Näätänen (1975) however points to the questionability of determining negativity against a pre-S1 baseline which, through its own fluctuations, may permit no CNV to be detected. Such a 'resident' CNV is considered by Donchin et al. (1975) and Wilkinson (1976) when subjects must maintain a preparatory set over a long period, but lacking experimental demonstration "remains moot" (Donchin 1977). In a review of the experimental evidence, Tueting and Sutton (1978), whilst not being able to unreservedly support formulations favouring either complete independence or a partial, but independently expressed, common source for the CNV and P300, did come down against any strict relationship on the basis of dissociation and non-proportional changes.

Different topographical distributions of simultaneously recorded CNV and P300 are also reported by Poon et al. (1974), Marsh et al. (1974), and Peters et al. (1977) with the former wave showing a maximal amplitude at the vertex and the latter parietally. Dissociation and nonproportional changes have been demonstrated in a variety of paradigms. Donald (reported in Karlin 1970) found that EPs during sustained CNVs were similar in amplitude to EPs in the absence of CNV. Whilst Cohen (1969) notes the tendancy for subjects who have "high positive amplitudes " (probably, in this case, N1-P3) to have large CNVs recorded in separate paradigms, this does not always hold true, i.e. some having large P300 amplitudes do not have large CNVs and vice versa. In a CNV paradigm, P300 amplitude significantly correlated ($r=0.43$) with ISI negativity. No statistical associations were reported between any latency or amplitude measure of the auditory or visual evoked potentials and the CNV are reported by Small and Small (1970). However their

use of a 0.5 sec. ISI makes it improbable that their CNV measures reflect a mature waveform. LPC amplitude is reported enhanced in a guessing paradigm experiment both with incorrect over correct guesses, and in acquisition rather than overtraining phases; these changes being independent of CNV at each recording site (Marsh et al. 1975 Poon et al. 1974). The latter also report significant correlations between P300 amplitudes during acquisition, but not CNV, and internal-external locus of control; high internal subjects having a larger LPC than externals. Accordingly, both conclude that these two electrocortical phenomena reflect quite different processes. Whilst noting that it implied no causal relationship (Donchin et al. 1976a) reported that CNV and P300 areas and interaural distribution varied in much the same way as a function of task complexity in a numeric sequence guessing paradigm. Donchin et al. (1975, 1976b) reported that P300 amplitude was determined neither by the amplitude of preceding CNV nor by the presence or absence of a preceding warning stimulus. Whereas a warning stimulus stimulated the development of a CNV, changes in P300 amplitude reflected independently changes in stimulus probability. They concluded that the P300, as an independent phenomenon, is an endogenous cortical component involved in information processing requirements of the subject's task rather than by pre-stimulus, diffuse, generalised, preparatory activity. The differential responsiveness of the P300 and CNV is supported in a reaction time experiment (Peters et al. 1977). Irrespective of response requirements, the CNV was recorded maximally frontally and the P300 parietally. The introduction of a response requirement led to a larger CNV at all electrode sites, but not to increased P300. In a separate paired-associate learning experiment, CNV amplitude decreased across presentation blocks at all recording sites, whereas P300 progressively increased.

Donchin et al. (1976b) and Smith (1977) note the possible confounding effects of CNV resolution on the P300.

Attempts have been made to separate these. Donchin has repeatedly extracted components, which he interprets as indicating the separation between the two, through the application of principle component analysis. Using implanted electrodes with a small psychiatric population, Papakostopoulos and Crow (1976) introduced, on a quasi-random basis, a tone into the ISI which instructed subjects to abandon the MR to S2. P300 was in no situation recorded anteriorly, although posterior recording detected both CNV and P300. A prominent positive component followed the imperative stimulus irrespective of whether an interpolated stimulus had been presented or not. CNV resolved to baseline in those trials when an interpolated stimulus was presented with its presentation. They concluded that P300 is separate from the CNV. Finally, Roth et al. (reported in Callaway 1975) used a task which varied in complexity so that the response could be delayed. CNV resolution was found to correlate with response latency, but P300 development occured independently of this.

In conclusion, that the P300 and CNV have different topographies, that either can be elicited without the other, and that no significant covariance between them, either directly or through an mediating variable, is consistently reported suggests that they reflect predominantly independent psychological processes. However, the present author has been unable to locate investigatory studies into possible P300 relationships with components of the CNV using long ISIs.

1.3. The P300.

The first report on the P300 (Sutton et al. 1965) coincided closely with the initial publication on the CNV (Walter et al. 1965). Although the CNV gained more research attention initially, the position is now reversed and the P300 is being subjected to intensive and extensive investigation. However attempts to establish a unifying theme or theory for this electrocortical event have proved abortive. As existing experimental paradigms were modified and the P300 observed in different situations so inconsistencies in its latency, topographical distribution, and responsiveness between individuals to task demands suggested that the then current descriptive models were inadaquate. Picton et al. (1978) produced a very readable review of the main issues, as has Sutton (1977) of psychological considerations involved. Other comprehensive review articles may be found in Donchin et al. (1978), Tueting (1978), and Pritchard (1981). Recent conference reports, including extensive reporting of experimental results and theoretical issues, are to be found in Otto (1978), and Kornhuber and Deecke (1980).

I have referred to the P300 as though it were a multi-faceted, yet commonly labelled, phenomenon. That the situation is not so elegent is summarised by Roth (1978). He notes that, of the three criteria (voltage, latency and location) commonly adopted to identify and separate components, location permits at least four anterior-posterior distributions to be specified. First, is a predominantly parieto-central distribution characteristic of most events in the 'P3b' range and missing stimulus potentials. Second, a predominantly fronto-central waveform (P3a) derived in stimulus change, in response absence, and with rare non-target, but task relevant, visual stimulus paradigms. Third, a parietal-occipital wave (P4) related to feedback and having a longer latency of around 650 msec. Finally, a slow wave which is positive in parietal derivations, neg-

ative in frontal, and almost absent at the vertex. Different researchers have adopted different conventions to describe P300 phenomena. Currently vogue are the following terms; P300, P3$\bar{0}$0, P3 or P3 component or P3 wave, event-related positivity (ERPOS), late positive component or wave or complex, P3a and P3b. Tueting (1978) observes that separate phenomenon may be proposed on the basis of experimental paradigms of topographical distribution, e.g. P3 no-go or P3 vertex. Uneless otherwise indicated, with the exception of the P3a, the present author intends to treat these terms as synonymous and they appear in the text as labelled by the researcher concerned. Only the P3a and P3b variants will be separately discussed.

Before turning to the responsiveness of the P300 to experimental and subjective manipulation, certain general characteristics may be introduced. The P300 is a positive-going peak, recorded maximally parieto-centrally, with a latency of about 300 msec that can be modulated in amplitude by variations in the psychological context in which the stimuli are presented. It commonly occurs in conjunction with the detection of a target stimulus, which must be regarded as task relevent, i.e. prompt a special cognitive or motor response. It follows a point in time when relevant information becomes available to the subject, regardless of whether that point in time is signified by a special stimulus or not. Its amplitude reflects trial-by-trial variations in the subjective probability that the experimentee ascribes, not necessraily consciously, to the event. It is faithfully elicited in a wide range of paradigms, e.g. simple, disjunctive and discrimination R.T. paradigms, feedback in guessing conditions, and stimulus matching tasks, which suggests that more than attentional processes, as suggested by Rösler (1980), are involved. In comparison to P3a, P3b has a longer latency, higher amplitude and a more posterior distribution. As such it may, or may not, be equivalent to the P300 originally described by Sutton et al. (1965). It is possible to propose that the P300 serves

an important adjunct to human information processing because it seems related to stimulus evaluation time and is independent of response selection and execution processes (Donchin et al. 1978; Picton et al. 1978; Tueting 1978; Hillyard and Picton 1979; Picton 1980). Its amplitude is put at 10-15 μv. (Rösler, 1980). A possible exception is the positivity elicited by response inhibition in a "go/no-go" task, which has a more frontal distribution (Donchin et al. 1978; Tueting 1978; Picton et al. 1978).

Improbable stimuli can occassionaly elicit a late positive wave even when irrelevant and unattended (Roth 1973). This 'P3a' is elicited by low probability auditory stimuli presented while the subject is engaged in another task (Picton et al. 1978; Tueting 1978) or by shifts in stimulus intensity in either attended or unattended channels (Picton et al. 1978). It has an earlier latency (around 240 msec) and is frontally maximally recorded. N.Squires et al. (1975) demonstrated P3a in response to shifts in stimulus pitch or loudness (latency: 220-280 msec), the independence of which was substantiated by principle component analysis. It has been described as the psychological correlate of "noticing but not paying much attention" (Picton et al. 1978) or "passive attention" (Tueting 1978) and linked with the orienting reaction in that it is elicited in the same situations and by the same variables that elicit the classical autonomic components of the orienting reaction (Friedman et al. 1978; Rösler 1980). The frontal P300 elicited by novel stimuli, with a latency of 360-380 msec (Courchesne et al. 1975, 1978), and reported below, is suggested to be a better candidate than the P3a as an index of orienting by Tueting (1978).

Certain evidence is presented as demonstrating that P300 is an endogenous event, such that Tueting (1978) asserts that "the existance of an independent P300, related to specific cognition and stimulus identification, is no longer the issue". P3 wave is elicited in a variety of decision-making tasks in which the physical characteristics

of the eliciting stimulus seem irrelevent to its appearance so that virtually identical distributions are obtained across the scalp to auditory, visual and somatosensory stimuli. As such it is not modality specific (Hillyard et al. 1978; Tueting 1978; Shagass 1976). The P300 can be independent of the presentation of a physical stimulus (Sutton et al. 1967; Donchin et al. 1978) and is emitted by an apparently internal trigger if stimulus absence is both significant and time-locked by some time-marking event (Tueting 1978). Picton (1980) states that such ERP components reflect as much the subject's cognitive approach to sensory information as any underlying psychological principle. Accordingly, amplitude is increased by such factors as task difficulty and subjective certainty (Picton 1980). Variability in its latency indicates that it occurs post-stimulus identification and responds to difficulties in evaluation (Donchin et al. 1978). The emission of P300, as a post-stimulus evaluation event, is more fully discussed below. Pritchard (1981), summarizing the prevailing ethos, states the P300 reflects only the general information properties of stimuli, i.e. its task relevence and degree of improbability. Neither the specific sensory information carried by the stimulus, nor the type of response required, seem to be reflected. The independence of P300 from the physical characteristics of the stimulus has been questioned recently in an experiment by Roth et al. (1980b). Varying auditory stimulus intensity in three conditions of count, ignore, and engage in another task, they reported increasing amplitude with increasing intensity of stimulus and as a function of task. With the highest level of stimulation (115dB) amplitude reached 20 μv. in the count condition, 16 μv. in the ignore, and 14 μv. during engagement in the other task. At 55dB the amplitudes obtained were 5 μv. in the count condition and 0 μv. in the other two. Intermediate levels of stimulation obtained a corresponding P300 amplitude. Latency, distribution, and sensitivity to task demands and probability all identified this waveform to be

the P300 and distinct from the P200; it was further seen to be free from artifactual contamination. Autonomic variables, as a source of interference, may be discounted due to their later onset latency than most ERPs.

Functional equivalence may be the common denominator linking P3bs, but their separation from P3as on the basis of topographical analysis raises the question of separate neuronal generators. This introduces two issues; firsty, to what extent the P300 is unique, and secondly, what do its various manifestations have in common. Taking the first question, two modes of attention are posited corresponding to Broadbent's (1971) stimulus and response set. N1 amplitude and its enhancement to stimuli in the attended channel is suggested to reflect the stimulus mode (see Figure 1). Incoming stimuli are selected for analysis and evaluation based on simple stimulus characteristics. Response set, involving P300 emission and enhancement, reflects terminal aspects of the subject's judgement about stimulus properties, and such a perspective is supported by the effect of varying the content of the eliciting stimulus. The P3 wave, in this hierachical model represents some aspects of target selection within the attended channel (Ford et al. 1973; Hillyard et al. 1978; Picton et al. 1978; Hillyard and Picton 1979). Hillyard et al. (1978) argue that such a premise is supported by various P3 characteristics; namely, by late onset latency, by equivalence of simple and complex cues in its emission, by its independence of high or low rates of stimulation and of the stimulus's physical properties, and by its enhancement by low probability stimuli. Evidence both for and against such a proposal may be cited. Smith et al. (1970) found that P300 amplitude was equal and enhanced in both attended and unattended channels to target clicks. However, ascertaining the degree of subject compliance with instructions to 'ignore' are more problematic than those of 'attend'. By contrast, Hink et al. (1978), again in a dichotic listening task, reported selective N1 enhancement only in the attended

channel (male or female voice), and P3 wave enhancement only to the target stimulus in the attended channel without further increment to the N1 component. K.Squires et al. (1973) report that, although highly correlated with each other, N1 and P3 are related to information processing in fundamentally different ways. The N1 component may be taken to relate to sensation level in a remarkably monotonic fashion (Roth 1978). However, Tueting (1978) notes that this component is not always present. She posits its non-appearance in the EP may be due to its being obscured by other post-stimulus events which compound its small amplitude and, assumed, latency variability, or that it may not always be elicited; the dependent subjective and other variables determining its presence having yet to be clarified.

Roth (1978) reports on the second question, as to whether P300 phenomena represent a single or divisable neuronal process. He notes that different distributions, just as for different amplitudes and latencies, need not imply different neuronal generators. Similarly, available evidence does not permit categoric statements in favour of a single generator, which is manifested with different latencies and topography. That latency differences cannot be taken as irrefutable proof of separate processes is indicated by experimental results demonstrating latency variation in response to variation in stimulus complexity as, for example, from Kutas et al. (1977), Kutas and Donchin (1978), McCallum (1980), and Perrault and Picton (1980). Näätänen (1975) and Tueting (1978) suggest that late positivity may represent a single phenomenon which is truncated in latency, at frontal electrodes, by the slow negative wave, and lengthened posteriorly by the slow positive wave (see Figure 1). This suggests that P300 anterior-posterior amplitude varies with task demand, and is supported by the absence of any definite peak in some experiments. A P245, possibly identifiable with the P3a, is reported by Vaughan et al. (1980). On the basis of interaural topographical anal-

ysis, they report this as generated by sources in the primary and secondary auditory cortex within the superior temporal plane and lateral surface of the superior temporal gyrus. Later positivity was noted but not systematically examined.

The comment of Donchin (1977), that the current task is one of establishing the psychological function of ERP manifestations of neural activity, has already been noted above. Sutton (1969) describes ERP studies as unique in that they involve simultaneously three types of event - physical, psychological, and physiological. He stresses that it is not sufficient to concentrate on only two aspects. Particularly the subjective element must not be overlooked as subjects usually have a diffrent perspective of the experimental situation than that given by instructions, i.e. differently perceive the link between physical and psychological events. It is the influence of this "subject option" that necessitates instructions to be clear, precise, easy to comply with, and making subjective sense. He suggests that reports that the later components of the EP, which relate particularly to complex psychological state, are 'labile' have not properly controlled for such aspects in the experimental situation. The plethora of explanations for late positivity, obtained under the same or different, experimental paradigms testifies to the lack of any comprehensive theoretical model. Outlining the differences between correlational and theoretical explanations in science, Pritchard (1981) suggests that P300 studies exemplify the former. The very range of variables manipulated itself mitigates against theoretical explanation. One consequence of this lack may be the attempt to establish logical functional boundaries that differentiate P300 activity. Rösler (1980) argues that determinants of ERPOS cannot be derived simply from behaviourist analysis of stimulus-response parameters, nor from the whole context of the experimental situation, but rather by theories about the different stages of information processing, including ex-

plicit formulations of subjective expectations.

In the following sections, experimental reports will be presented broadly separating emphases on stimulus information characteristics from subjective or task relevance factors. Within each section key factors are presented, together with, where possible, alternative explanations. Thus, for example, a P300 eliciting stimulus may serve to reduce uncertainty or through its complexity or presentation-probability deliver information. Very broadly, the two sections outlined here may be described as concerned with information theory and with subjective or cognitive approaches. The application of such descriptors is contested by those arguing explanation in terms of arousal processes, which will also be discussed. Finally, studies will be presented reporting on the significance of latency. It must be stated though that this review is not exhaustive, and such reports that are included are presented to illustrate the conceptual and experimental field to date.

1.3.1. Variations in informational content of stimuli.

In the first report on the P3, Sutton et al. (1965) observed that amplitude was enhanced when subjects had to guess what type of stimulus would be presented in comparison to when they received prior information. Also, stimuli of low probability elicited higher amplitude P3 than higher probability stimuli. This result, and a second series of experiments (Sutton et al. 1967), demonstrating that P3 occured when the second of a pair of tones was, and was not, presented, suggested that the eliciting stimulus, or its absence, allowed for the resolution of uncertainty. The P3 was interpreted as related to the "effective information content of the stimulus". Donchin and Cohen (1970) reported a large positive wave in the ERP in response to counted stimuli of a latency of around 200 msec, although in later reports this latency was reported as about 250 msec (Donchin

1977) and between 200 and 300 msec (Donchin et al. 1978); as has already been suggested P300 latency has some bearing, at the very least, on the eliciting stimulus. P300 was seen to be dependent on some combination of uncertainty resolution and task relevance (Donchin 1977).

Tueting et al. (1970) conducted a further series of studies on the objective, or a priori or experimenter-given, probability. In the first experiment the target stimulus, of two stimuli of a combined probability of 1.0, was presented in a block of trials with 20%, 40%, 60% or 80% probability. In the second experiment, although the overall probability within blocks was 50:50, the sequential probability of target stimuli following each other was manipulated to give repetition probabilities of 20%, 40%, 60% or 80% within a trial block. Both experiments were presented with subjects having prior knowledge (certain) and having to guess (uncertain) the coming stimulus. In the uncertain condition, relative frequency (in the first experiment) and both alternation and repetition probability (in the second) showed a clear monotonic relationship to P300 amplitude and probability for both 'hits' and 'misses'. However, relative and alternation frequency both demonstrated a significant relationship in the certain condition; the former association being replicated by Friedman et al. (1973). This is not compatable with information theory because, since subjects knew the coming stimulus, then its delivery carried no information. The status of repetition probabilities' association with P300 in the certain condition remains anomalous. Accordingly, the formulation that the nervous system responds to novelness per se was postulated. Nonetheless, unpredictable events are more important than predictable, although prior knowledge is not equivalent in all its forms. Thus, a "novel change" is more critical than a "novel repetition" so that events with a greater redundancy produce a smaller response (Sutton 1977), e.g. simple alteration as against longer stimulus chains. Tueting (1978) suggested that repetition effects could be related to cognitive

variables such as the subject's running calculation of absolute and sequential probabilities of events and the calculated risks involved in making one guess rather than another.

Various studies, investigating the effect on P300 amplitude of a priori variations in stimulus probability (this not necessarily being the same as subjective probability) have as a general conclusion the observation that amplitude varies inversely with probability (Donchin 1977). Donchin et al. (1978) varied the comlexity of a sequence generating rule in different series of trials, instead of manipulating the a priori probability. Stimuli were either alternated, or presented randomly, or were presented in a sequence of nine stimuli, which subjects had previously memorised or had to infer from the series. Smaller P300s were reliably associated with the more predictable stimuli. This relationship was systematically demonstrated by Duncan-Johnson and Donchin (1977) in an 'oddball' paradigm, in which subjects are presented with a Bernoulli series of stimuli and are instructed to attend in some way to one distinctive event. In their experiments, subjects were instructed to count tones of a particular pitch. Whether higher or lower tones were counted the results demonstrated an inverse relationship between P300 amplitude and probability over its entire range. Further, at every level of a priori probability, the magnitude of the P3 complex was reduced when the eliciting stimulus repeated the preceding tone, and was enhanced when it was preceded by the other tone. The relevance of the task was underlined in that if subjects ignored the tones and engaged in some other task, e.g. reading a book, no P300 was obtained. In a further, and modified, form of this experiment (Duncan-Johnson and Donchin 1978) a Bernoulli sequence was presented in a S1-S2 paradigm in two conditions. In one, S2 could be either of two stimuli, and in this condition P300 to eliciting stimuli developed according to the incremental probability indicated by the increasing length of preceding alternative stimuli.

In the second condition, S1 either indicated which of the two stimuli would be presented as S2 or was a third stimulus indicating that they were equiprobable. ERPs were greatly reduced when the warning stimulus indicated a priori probability. Johnson and Donchin (1978) demonstrated that P300 amplitude on any trial is closely related to the [illegible] stimuli presented in the preceding Bernoulli [illegible]. Reduction in P300 reflected the accumulated improbability of the irrelevent stimulus in the preceding chain. The same effect for visual stimuli was demonstrated by K.Squires et al. (1977). Donchin (1977) suggested, accordingly, that the P300 manifested processes that occur after stimuli have been categorised according to physical properties into classes determined by those stimuli.

Studies investigating the effect of probability require that expectancy, being a determinant of the P300, be held constant whilst task relevence of the presented stimuli is varied. Two approaches to control expectancy have been applied. Subjects can be presented with two stimuli which are independently varied and to which the subject selectively responds, or alternatively, situations in which the subject ignores all stimuli and engages in another task, which is unrelated (Donchin et al. 1978). Representative of the first approach is an experiment of Donchin and Cohen (1967) who varied either the figure or the ground of a visual display, with one such variation being relevent in different series. Only that aspect designated as task relevent elicited large P300s. An unpublished study by Duncan-Johnson et al. (reported in Donchin et al. 1978) investgated the effect of simultaneous auditory and visual presentation of stimuli. Subjects were required to count rare stimuli in either or both modalities. Rare auditory and visual stimuli elicited similar P300s when both modalities were counted, but the same stimuli failed to elicit a P300 when its modality was attended and stimuli not counted. Such was interpreted as due to the effects of selective attention. Basic to any such investigation is the degree to

which rare or unexpected stimuli intrude on the subject's attention. In a passive condition it is difficult to ascertain to what extent subjects ignore the irrelevent channel. P300 enhancement to rare and ignored tones is reported by Ritter et al. (1968). Smith et al. (1970) dichotically presented different series of numbers, letters and clicks. Subjects were instructed to attend to one ear or the other and report either the clicks or the letters. Whilst monitoring the clicks in one ear, the click-emitted P300 (latency: 200-300 msec) in both ears was enhanced; there being no difference between attended and unattended ears. Also, interestingly, P300 was of higher amplitude to clicks than letters. Courchesne et al. (1975) presented subjects with a numeric target figure of a "4" to be counted against a background, not to be counted, of "2"s and intrusive material of geometric figures and unrecognisable 'novels'. Counted stimuli produced a parietal P3 (latency: 380-430 msec), whereas novels elicited large frontally distributed P3s (latency: 360-380 msec). With repeated presentation of novels maximal recording shifted from Fz to Pz (Courchesne et al. 1978). Selective attention was presented as an alternative explanation to their posited information processing of rare stimuli (Rösler 1980). He objected that probability experiments have failed to take into account the intrinsic rareness of irregular target stimuli. Frequent non-target stimuli are processed automatically, whereas rare stimuli, both target and non-target, which cannot be processed automatically evoke ERPOS. However, this objection is already noted by Courchesne et al. (1978) who concluded that P3 waves may be affected by the ease with which events can be categorised, their subjective importance, their a priori probability, and by sequential event structure. However, such experiments do illustrate the impact of the subject option, such that their expectancy may not follow a priori probability: reference, for example, the obtained differences between novels and geometric figures. While improbability, objective or sub-

jective, may be a necessary condition to elicit P300s, it is not sufficient. Clearly involved , along with probability per se, is the distinctive function any stimulus has for the subject.

Turning attention to experiments controlling attentional or expectancy effects through the introduction of a second task, already noted is the abolition of P300 while subjects worked on a word puzzle (Duncan-Johnson et al. 1977). Wickens et al. (reported in Donchin and Israel 1980), by introducing a second task of monitoring cursor movements, attempted to assess the demands of the first (counting targ[illegible]li in a [illegible]) and to counted stimuli was reduced with the introduction of the second task; the reduction being the same whether cursor movement was tracked in one or two directions. This was confirmed by Isreal et al. (1980a) who observed, after the [illegible] reduction with the introduction of the tracking task, that no further reduction occured as the speed of the cursor movement was increased. In a further report, (Isreal et al. 1980b), they demonstrated that, when subjects have a task of monitoring a visual display with the [illegible] counting auditory stimuli, P300 amplitude decreased as the number of visual targets increased. Thus, the P300 can be seen to be sensitive to changes in perceptual demands even though it is insensitive to changes in task demands that are based on the motor attributes of the task. The results are very much in accord with those of Stuss et al. (1980) who concluded that, "the processing manifested by the P300 is limited by the available supply of resources that subserve perception". Donchin and Isreal (1980) warn against the invocation of 'subjective factors', as a qualification of task relevence, to account for residual variance in probability experiments in which the experimental setting of a priori stimulus presentation has not been controlled. Thus, failure to contain fluctuations in expectancy, which is the reciprocal of probability, in some experimental designs should not permit the basic

responsiveness of P300 to objective probability to be refuted.

1.3.2. The effect of subjective or task-relevance [illegible]

Hillyard et al. (1978) and Hillyard and Picton (1979) posit that the amplitude of the P3 wave is determined both by the confidence of detection and by the subjective liklihood of stimulus presentation. These have been studied by investigating subject's confidence ratings and using stimuli following guesses as to the coming stimulus, and are introduced below. Such considerations are necessitated by the inability, even in controlled studies, to account for total variance. However, it is necessary to adopt a wider than single trial, or successive trials, perspective. As Donchin et al. (1978) note, task relevance needs to be evaluated in terms of the subject's overall task. Information that is required for successful task execution is carried by the stimuli that the subject may or may not process or perceive (Hillyard et al. 1978; Hillyard and Picton 1979). Any stimulus gains its relevance from the extent to which the subject can use it to increase success in overall performance. Thus, both strongly held hypotheses, that are disconfirmed, and any of equally strongly held hypotheses, that are confirmed, all produce P300: such a formulation being prompted by the experimental observation that infrequent events can fail to elicit a P300 and that frequent events can elicit it. Accordingly, the individual is seen to be continually interacting with the environment, rather than a specific response selection procedure activated on any particular trial. Any stimulus, once identified and categorised, initiates two processes; one being concerned with response selection, and the other with 'conceptual updating' (Pribram and McGuiness 1975).

Subjective probability, being a major determinant of P300 amplitude in certain circumstances (Donchin 1977), has

been manipulated by instructing subjects to guess the coming stimulus. Attempts have been made to determine whether stimuli which confirm or disconfirm guesses elicit different ERPs. Sorting the data of Duncan-Johnson and Donchin (1978), according to alteration or repetition prediction demonstrated that alteration predictions, that are confirmed, and repetition predictions, that are disconfirmed, both elicit large P300s. However,as Donchin (1970) observed, such results are amenable to the alternative interpretation that stimulus alterations, irrespective of subjects' prediction, produce larger P300s. Thus rarity, and its suprise value, rather than feedback value could be of central importance. The same assessment can be applied to the results of Friedman et al. (1973), who demonstrated the dependence of amplitude on the relative probability of feedback stimuli confirming or disconfirming guesses.

Other studies have also attempted to demonstrate that the major determinant of amplitude of the late positive component is the probability of the response to the stimulus rather than the stimulus probability itself. The alternative explanation of increased P300 to stimulus rarity (Donchin 1977), already noted, is questioned by results obtained by Friedman et al. (1978). They demonstrated that [illegible] signals reflected their cumulative improbability, i.e. was a function of the class of improbable events. Picton et al. (1978) reported that the amplitude of the late positive component depended on the task relevent information in the feedback stimulus as measured in bits as -log2. Cambell et al. (1979) concluded, on the basis of a series of experiments, that the feedback P3 component varies with the improbability of that stimulus and is unaffected by its meaning. Their second conclusion stated that P3 amplitude is closely determined by contingent probability, which is the probability of a stimulus given a particular response; the response a subject makes [illegible] the perceptual set or expectancy prior to receiving feedback. It has been suggested (Rösler 1980) that such a

result could be artificiallly obtained by recording during different cortical states. He argues that the value of feedback lies in its congruence or incongruence with subjective expectation. Developing a mathematical formulation of a priori probability in interaction with subjective expectations and their confirmation or disconfirmation, he obtained hypothetical amplitudes that closely correspond to those actually reported by Cambell et al. (1979).

Confidence in signal detection has also been associated with increased P300 amplitude; this relationship describing both 'hits' and 'false alarms'. However, with increasing signal detectability, P300 amplitude was reduced irrespective of confidence ratings (K.Squires et al. 1978). Thus both confidence and probability of signal detection selectively affect P300 amplitude. Confidence is seen to reflect, a posteriori, subjective uncertainty of having correctly perceived an event; it is thus inversely related to equivocation. Resolution of such uncertainty is reported to determine P300 amplitude. Sutton (1977) reported that evoked P300 amplitude, having corrected for latency variation, was larger than for emitted P300 amplitude; that is, the delivery of a stimulus allowed uncertainty to be clearly and sharply resolved. Accordingly, the information received by the subject determines P300 amplitude. As suggested by classical information theory, the ammount of information received by the subject is determined by their a priori uncertainty over an event's occurence minus an information loss (equivocation) due to their uncertainty over having correctly perceived it. Thus, a rare stimulus in an unattended channel will only add to P300 amplitude when it contributes to reduce a priori uncertainty (Rushkin and Sutton 1978a). In an experimental report (Rushkin and Sutton 1978b), subjects had to estimate a time interval (700 or 1500 msec) at the end of which a stimulus was, or was not, presented. The report of lower emitted P300 amplitude was reconfirmed. This was more marked with the longer interval and was attributed to higher temporal uncertainty

in time estimation.

P300 has also been elicited by completely predictable events. Friedman et al. (1975) demonstrated that, when words were presented in sentance structure, the word at the end was associated with a large P300. Voice sounds also produced large P300s, which was attributed to their non-appearance in everyday speech; this may be on a par with positivity to novel stimuli as reported by Courchesne et al. (1975, 1978). By contrast, these results were observed at all electrode sites and for all subjects. That words elicited P300s was associated with 'syntactic closure' and the smaller P300 amplitude attributed to voice sounds, not to the associated probability, but to their inclusion in a class of non-signal stimuli. Alternatively, it may be suggested that higher amplitude to words are explicable in terms of their malleable semantic meaning. Picton et al. (1978) report that stimulus meaning, which varies with the context in which it is perceived, may also affect P300 amplitude. They report that when stimuli were ignored, while subjects read, or when non-stimuli were counted or provided performance feedback, they elicited ever larger late positive waves although their objective probability, and hence information content, remained the same. Sutton (1977) applies the term 'salience' to describe how the meaning of the stimulus in the experimental context is of relevance to the subject.

Although decision processes in different tasks are undoubtably different, a coherent theme could be provided by comparisons against a memory template, as suggested by Ritter and Vaughan (1969) and K.Squires et al. (1973). According to an orienting perspective, low probability and novel stimuli would represent mismatches. These would require extra time for comparison over matches which occur earlier in latency and of higher amplitude (Posner et al. 1973; Thatcher 1977). Tueting (1978) observes that such a model does not contain results from probability experiments which demonstrate lower P300 amplitude in

association with high probability (match) events. She suggests that this paradox can be resolved by postulating two match-mismatch processes; a frontal P300 for probability, and a parietal for more complex decision tasks. Low probability events are inherently more significant to the organism than high probability familiar events, even if they are not designated as targets by instructions.

Poon et al. (1974) investigated the LPC, as reflecting information processing, and internal-external (IE) locus of control (Rotter 1966). Internal control refers to the perception that positive or negative events are personally controlled, and external to the expectancy that events are unrelated to personal behaviour and beyond personal control. On the basis of a binary-choice gambling paradigm, they reported higher LPC in acquisition trials, when subjects were attempting to learn the stimulus sequence, in comparison to overtraining, when the solution had been obtained. Further, the more internal a subject, as categorised by IE score, the larger was LPC amplitude during acquisition trials; this relationship being significant at parietal and vertex recording sites. During acquisition 'internals' gambled conservatively which suggests that 'confidence' per se cannot account for their higher LPC amplitudes. Further, with overtraining, all subjects were betting the maximum but, in comparison to acquisition trials, LPC amplitude had declined.

1.3.3. The explanatory value of arousal processes.

Attention is defined as the process underlying the selection and organisation of available information, whatever its source, for appropriate response, both overt and covert (Picton et al. 1978). It is intimately related to general arousal, which controls the availability of both information and response. In the signal detection paradigm, target stimuli enhance the N1 component and elicit a large

positive component suggesting that two levels of selective attention are in operation; corresponding to the stimulus and response set defined by Broadbent (1971). They cite in support experiments by Hink et al. (1978) and Courchesne et al. (1975), which have already been noted, as well as their own dichotic listening study. Tone stimuli were randomly allocated to one or the other ear, presenting different intensities on delivery. Subjects were instructed to attend one ear and to detect target tones of a third, and lower, dB. N1 amplitude was enhanced to the attended ear relative to the unattended, and the target stimulus elicited a large positive wave between 350 and 550 msec These changes they interpreted as reflecting stimulus and response modes of attention. While the P300 may well be influenced by arousal level, its equivalence to response set attentional processes has been questioned. Näätänen (1975) contradicts the notion of the P300 being a code for selective attention due to its occassional longer latency than the associated behavioural event. His (1973) objection to the citing of attentional processes, on the basis of correlational studies, has already been noted in the preceding discussion of arousal involvement in the CNV.

Karlin (1970) reviews the alternative hypotheses of differential preparatory arousal and reactive change following stimulation to account for results reporting enhanced EPs, i.e. as due to non-specific changes. He objects to reports of enhancement in an attended channel as due to selective attention because such effects could be as easily described by a selective preparatory arousal in that channel. Unfortunately for such a hypothesis, rare events have been found to elicit higher amplitude P300 than more frequent. As he notes, anticipatory differential arousal requires than sequences between stimuli need to be predictable and the interval between them long. The question remains as to whether reactive changes in arousal offer a defensible alternative. Karlin and Martz (1973) compared different responses to rare and frequent tones, following a

warning light, enabling the comparison of the effect of both stimulus and response on the P300. Neither variable alone was significant, but combined significant results were obtained, i.e. rare stimulus plus rare response versus frequent stimulus with frequent response. Correcting for differences in negativity in a condition delaying response allowed significant differences between frequent and rare responses to emerge. They suggested that enhanced P3, in the rare stimulus-rare response condition reflected changes in phasic arousal, as well as possible cognitive factors. The results of Duncan-Johnson and Donchin (1977) are not so amenable in that responsiveness of P300 amplitude to objective stimulus probability requires the supposition of graded changes in arousal.

Näätänen (1967) demonstrated that EPs to clicks presented during the foreperiod were greater than those presented between trials, so suggesting a post-warning stimulus differential arousal. Such a demonstration would need to be extended to a comparison of EPs in different conditions of stimulus rarity before P300s can be accounted for solely in terms of differential arousal. What is interesting is that the 'inside' clicks were presented, randomly over trials and time point within trials, only 39% of trials whilst 'outside' occured in 44%. Karin's suggestion that internal clicks provided additional warning markers is dismissed by Näätänen (1975) arguing that introducing the stimulus whilst subjects await the target stimulus involves discriminating the degree of relevence, a posteriori, of the interpolated stimulus to the experimental task. As such, it would appear to be a comparatively rare event to an on-going task, i.e. subjects would have to discriminate between it and the target stimulus. It may also be noted that probability theory in itself suggests that lower probability signals elicit higher amplitude P300s, as obtained for the inside clicks. Callaway (1975) suggests that uncertainty may be equated with arousal.

In a further experiment (Näätänen, 1967), differential

preparation was precluded by randomly embedding targets among others of the same modality and randomly mixing visual and auditory stimuli at the same time as varying the ISI. He reported that no differences between target and non-target clicks were observed. However, in this experiment P2 or P3 was enhanced to flash stimulation (Näätänen 1975). Wilkinson and Ashby (1974), in a replication of this experiment, found that P300 to auditory stimuli was affected both by stimulus relevence and probability.

Although accepting the non-applicability of differential preparatory arousal to account for the findings of Sutton et al. (1965), reporting enhanced P300 to rare events, and of Sutton et al. (1967), reporting evoked and emitted P300s to target stimuli, Karlin (1970) postulates that the reactive change model is appropriate. Accordingly, the positive deflection is a consequence of termination of a state of expectant readiness after the information processing activity itself. Against Karlin's reactive arousal hypothesis may be set the cognitive accounts of, for example, 'suprise' (Donchin 1977), 'contextual updating' (Donchin et al. 1978), or 'salience' (Sutton 1977). Rohrbaugh et al. (1974) used an iconic recomposition task, with horizontal and vertical lines forming S1 and S2, with ISI varying from 30 to 480 msec, to demonstrate that P300 was independent of reactive arousal processes. Although also applying a warning stimulus, 3 sec. before S2, preparatory arousal as an effective mechanism was seen to be contradicted by enhanced P300 in a random condition.

Näätänen (1975) summarises evidence that the P3 or P300 might, in many studies, indicate a non-specific change of state. Its occurence is too late to be a direct correlate of behavioural and psychological phenomena, its latency is highly variable, and often lacks any discrete peak. Against which may be objected that the P300 is not viewed as a discrete psychological event, however that might be measured, and is a process generally recognised to be independent of MR. Näätänen notes the possibility of a rel-

ationship with total negativity, even in the absence of a CNV, due to the presence of a pre-stimulation 'resident' negativity. Further, non-specific processes are seen to be indicated by the larger scalp topography than for other stimulus-locked responses, which is similar for both auditory and visual stimuli and for different tasks. However, such equally well may indicate non-specific cognitive processes. He suggests that some evidence indicates that the P300 could be a cerebral component of the orienting response. The argument is pursued that it may form a part of a much longer response pattern stretching from the P2, with which it can be confused, to as late as 400-500 msec post-stimulation. On this basis is the three component hypothesis of evoked potentials presented. Two successive peaks are included, referred to usually as N1 and N2, in addition to a temporally overlapping, longer duration positive process, which might have an onset latency as early as 50-100 msec, peak at 200-500 msec and may last up to one second. This positivity could peak before N2. Findings of Vaughan et al. (1980) suggest some support for this hypothesis. They report a frontally-maximal positive wave (P245) following the N1 which, on the basis of different topography, suggested multiple components between early and late segments. This early wave, having a possible identity with the P3a, demonstrated a rising positivity to peak between 300 and 350 msec However, this component responded anomalously in that it demonstrated higher amplitude to frequent than infrequent stimuli whilst subjects read. They also reported large amplitude P245 to the stimulus following a rare stimulus and which required a response. Also a small positive peak, parietally maximal, was only observed to tones following a frequent tone and requiring a response. Similar positivity was observed by Roth et al. (1980a) who reported a peak at the vertex of 220 msec and parietally of 260 msec in response to noise-burst stimuli. The skin conductance response had a much later onset (about 2 sec.) and declined much more markedly over the experimental

period. They concluded that the P300 varies independently of peripheral measures of arousal. Courchesne et al. (1978) have reported frontal positivity to novel stimuli, which has been related to orienting, which shifted to a parietal maximum with repeated presentations. However, frontal negativivity has also been associated with stimulus evaluation (K.Squires et al. 1973, 1975). Rushkin and Sutton (1978b) report reduced P300 amplitude in conditions of subjective uncertainty over information presented in stimuli. Such is opposed to the arousal hypothesis, which would rather suggest increased arousal with higher levels of uncertainty. As such the arousal hypothesis is neither proven nor disproven. Any attempts to identify cognitive factors as solely responsible need, though, to ensure that different states of arousal are not elicited by the very structuring of the experiment. Nash and Williamson (reported in Pritchard 1981) showed that P300 amplitude is sensitive to levels of arousal in a choice R.T. task. P300 in response to a probe tone immediately preceding a visual cue signal had a higher amplitude under instructions emphasising speed of response rather than quick but careful responses. The effect of instructions are also noted by Kutas et al. (1977) and Kutas and Donchin (1978) who stress, by comparison, cognitive factors. However, as Pritchard (1981) observes, demonstrating P300 responsiveness to cognitive variables does not mean that it is totally insensitive to non-cognitive factors.

1.3.4. Variability in latency.

It is traditionally problematic to measure ERPs which vary in amplitude, latency and topographical distribution to determine the uniqueness of any waveform. Whilst the last presents a distinct attribute, quantification of ERPs in terms of their magnitude is an, as yet, unsolved problem. Amplitude and peak latency are two separate, although

probably related, approaches to a description of the event itself: their combination in a single area measure tending not to be used due to the difficulty of excluding other ERP components. As already observed, latency measures can mark 'real' amplitudes, but evaluation of peak latency is also uncertain in that the variability of the event is as important as the mean of the event itself. As already noted also, there may be no distinct peak in the obtained waveform [illegible] timing of the event doubly problematic. Latency is reported in the literature as follows: 250-500 msec (Hillyard et al. 1978), 250-700 msec (Donchin 1977), 275-600 msec (Donchin et al. 1978), some 300 msec from the point of information delivery (Hillyard and Picton 1979), between 300 and 400 msec (Picton et al. 1978), and 300-500 msec (Shagass, 1976; Rösler 1980).

The variability and range of P300 latency, and the marking of the event advanced by Hillyard and Picton (1979) introduced above, indicates the fundamental issue. As Donchin (1977) and Donchin et al. (1978) observe, with endogenous components, the timing of the eliciting event, being internal, is unobservable and therefore cannot be measured. The only determinable event is an external one so that the internal processing which prompts P300 formation, must either occur at a fixed interval after it or be synchronised with it. There is no objective way of validating either of these alternative premises. The latency of the endogenous event is taken to depend on the time required to recognise the stimulus and to evaluate its feedback value information content, and both must contribute to P300 latency and perhaps account for its variability. Latency variations, thus, comes about because of the latency of the endogenous event which itself is dependent on the time required to recognise the stimulus (its 'discriminability') and to evaluate its relevance and subjective probability (its salience or meaning and subjective confidence in [illegible]

being response selection and initiation processes, seen in the overt response, and a stimulus evaluation process, indicated in the P300. Latency of P300 has been found to be shortest to a common name, intermediate for a class of names, e.g. Mary, Jane, and longest for word synonyms (Kutas et al. 1977; Kutas and Donchin, 1978). These results were interpreted in terms of stimulus evaluation time in preference to the resolution of uncertainty (Sutton et al. 1965). Similarly, McCallum (1980) and Perrault and Picton (1980) found latency to be increased in association with visual stimuli of increasing complexity. In simpler conditions the MR occured before peak latency, but after it with more complex stimuli. McCallum also observed protracted positivity in more complex situations. That P300 reflects stimulus evaluation time is further supported by its sensitivity to changes in stimulus discriminability, and to the absence of any relationship with R.T.

A review of the literature dealing with the relationship between latency and R.T. may be found in Tueting (1978). Whilst it is not the present author's intention to review this field, as it is incidental to present purposes, certain general observations are warranted. Tueting argues that whilst amplitude differences at a given latency may be identified, it is not permissable to argue that latency varies as a function of condition or electrode location. Additionally, variations in latency even within conditions can artificially produce lower amplitudes in the averaged waveform. That the amplitude of the later components would be increasingly reduced is indicated by the concomitant increase in variability with latency (Kutas and Donchin, 1978). Such would account for a report (Ford et al. 1976) that amplitude of P3 was reduced with increasing latency; these authors did not record standard deviation. Tueting (1978) suggests that examination of individual trials presents an alternative method of analysis. Applying a Woody filter to the data of Kutas et al. (1977) and Kutas and Donchin (1978) enabled correlations to be calculated for

each trial against R.T. The correlations obtained were lower for instructions emphasising speed (r=0.21) than accuracy (r=0.61); it should be noted that only few subjects were recorded. For P300 latency to vary systematically with complexity of information, subjects are required to comply with accuracy of response instructions rather than speed imperatives (Kutas and Donchin 1978) and this consequently increases R.T. (Donchin 1977). Further evidence on the independence of P300 latency and R.T., and both their dependence on the strategy adopted by the subject, is offered by a study by McCarthy (reported in Donchin and Isreal 1980). He presented subjects with the words 'right' and 'left' in conditions of upper and lower case with or without masking noise. Upper case required subjects to respond with the ipsilateral hand, and lower case with the contralateral. R.T. was found to be increased both by response incompatability and by the masking noise matrix. That the two effects were additive suggests that each affected a different, and presumably independent, stage of processing. P300 latency was delayed by 60-70 msec with the introduction of the noise matrix, but was unaffected by the manipulation of stimulus-response compatability. Donchin and Isreal (1980) conclude that P300 appears to be an index of stimulus evaluation time and independent of response selection and execution. Ford et al. (1980), on the basis of response time to probe digits to different memory set sizes, with and without the masking of digits to be memorised, suggests a parallel model which allows P3 and R.T. to separately mark the relative timing of the completion of the decision and motor responses. R.T. and P3 latency are both increased by memory set size suggesting that R.T. is affected by the processes occuring after P3. Probe digits, not in the memory set, were associated with a later P3 and R.T.; the increase being similar irrespective of masking. Increased latency to mismatch events is in accord with the match-mismatch model of Tueting (1978).

1.3.5. Functional interpretations of P300 activity.

It should be noted from the outset that the following accounts of the functional properties of the P300 are inferential in nature. They represent attempts to introduce a unifying theme to the disparate correlational studies. The justification for any postulated process, in the form of basic experimentation, having been reported above, will not be repeated here.

Although Karlin (1970) and Näätänen (1975) both argue that late positivity represents a reactive change in arousal consequent on sensory information processes, such is not sufficient to describe the P300 in all the situations studied. As a model, it relegates the subject to a role of passively enduring the machinations of the experimenter on a trial-by-trial basis. Global shifts in arousal, as for example by being in the experimental situation itself, are a matter of conjecture and are unamenable to experimental scrutiny. Donchin et al. (1978) present the P300 as a scalp manifestation of cognitive acts of quite specific properties which, although occuring in different tasks, has an invariant function in the sequence of information processing. Early functional accounts stressed the reduction of uncertainty (Sutton et al. 1965) in situations in which the P300 was, or was not, elicited, or passive information processing (Sutton et al. 1967). An informational perspective is limited in that signals differ in their task relevance as well as the subjective salience they present. Subsequently emphasis shifted to the examination of variables in P300 amplitude and latency. Accordingly, cognitive variables have been manipulated to investigate the information processing required of the subject. Stimuli that elicit P300 have at least two attributes in common (Donchin et al. 1978); these being, that they are unexpected, and that they have some relevance to the current task. Ritter and Vaughan (1969) conclude that P3 appears to be a

correlate of central processors for cognitive evaluation of stimulus significance. Shagass (1972) suggests that the P3 wave bears an intimate relationship to the orientation of the subject to the environment; and that attentive activity underlies all conceptualisations of its theoretical significance (Shagass 1976). An attentional process is also argued by Picton et al. (1978) and Rösler (1980). Various researchers suggest that expectancy on the part of the subject, reflecting certainty, is matched against incoming sensory information by a comparator mechanism, the output of which is manifested in the P300 (K.Squires et al. 1973, 1975; Ford et al. 1976; Duncan-Johnson and Donchin 1977). Expectancy is defined as suprise which is the reciprocal of certainty. A general activation of perceptual and motor responses to a significant event is postulated by Picton and Hillyard (1974) to account for P300 activity.

Elsewhere the meaning of the stimulus to the subject, or 'salience' (Sutton 1977) or 'suprise' (Donchin 1977), is emphasised. It has been interpreted to indicate the reaching of a decision (Rohrbaugh et al. 1974). Already noted is Tueting's (1978) suggestion that separate P300s may discriminantly represent match-mismatch processes. Noting that large P300s are elicited in guessing and discrimination paradigms and to low probability and novel stimuli, she suggests that stimulus information in such cases may influence future guesses, judgements or reactions. Stimulus presentation is thus concerned with the readjustment of templates in memory. Donald (1979) presented this viewpoint in his suggestion that the late positive component is a non-specific signal of a changeover in short-term memory processing itself, and as such possibly represents an 'erase' process occuring at the completion, or initiation, of such association cortex processing. Donchin et al.(1973, 1978) and Donchin and Isreal (1980) propose that the P300 reflects the activity of a general purpose processing system which is engaged whenever it is necessary to update the mental model of the environment; and hence including active

involvement on the part of the subject. Model updating is required whenever events occur the subjective probability of which are low and when occurence is relevant to the subject's current activity. Categorisation of an event must be complete, so that P300 amplitude reflects suprise value and its latency a measure of categorisation time. As such P300 indicates contextual updating, the modification of which is reflected in subjective performance and P300 activity in future tasks.

DEPRESSION.

2. Introduction.

An outline of depression is presented here so as to describe the clinical features together with a description of the division of the symptomatology into different disease states. A great deal of debate surrounds the number and definition of symptoms and, with increasing pace, contradictory classificatory schemes are proposed. Suggestions that the current confusion over depresssion is exaccerbated by such a plethora of schemas (Kendall 1977) are well taken. Depue et al. (1979) identify two sources for the modification of concepts of disease processes. The first argued that man's biological and psychological well-being was influenced by his physical and psychosocial environment, and the second, arising in the biomedical camp itself, stressed the genetic transmission of disorders. An appreciation of these arguments contributes to the understanding of concepts describing affective disorders.

From the medical camp comes the notion that homeostatic regulation of the body represents a dynamic regulation of constantly interacting and inherently unstable biochemical systems. Biologic variation within the tissue is introduced both by inherent metabolic activity and by interaction with the environment. Adaptive reactions, aimed at restoring and maintaining the dynamic steady state, is required through the activity of the cellular self-regulatory system, the external regulatory system involving the autonomic system, the neuroendocrine system and the immune system, and the external regulatory systems of the brain. Inasmuch as a dynamic steady state depends on the efficient operation of regulatory systems, any biologic predisposition to disease may be viewed as quantitative variations in malfunctioning normal body processes. In major psychopathological disorders, as depressive disease, the primary source of biological predisposition is likely to be genetic.

Another school of thought suggests that there is a contributory role in the development of disease from psychological predisposing factors which can provide a general setting in which disease is initiated. Thus, for example, dependent individuals respond to loss with a perception of personal threat leading to feelings of helplessness and hopelessness and the response of resignation. However this approach should not be too narrowly interpreted. That heterogeneous environments can serve as antecedents for disease development suggests the application of the biologic notion of a final common pathway; equivalent to the principle of equifinality in systems theory. Further, events per se are not of central importance, but rather is their perceived importance and the consequent emotional and biologic responses. This suggests that any final common pathway is cognitive in nature. Such a general psychological factor as cognitive schema may have the power to initiate disease due to its pervasive influence on appraisal and coping behaviour rather than due to any more atomistic idiosyncratic psychological characteristics. In conclusion, it may well be that the predictive power of a single predisposing factor is small. Consequently, presdisposition to disease needs to be assessed in a multidimensional framework, where several biologic and psychological variables are explored simultaneously (Depue et al. 1979; Goodwin 1974).

However, any predisposition, psychological or physiological, does not explain how a response is initiated in, for example, a disease process. Although the issue of quantitatively and qualitatively different response processes to different events is not yet resolved, appraisal processes and their concomitant emotions are likely to play a significant role in mediating the effects of the environment. The rationale for postulating emotional mediation is based on the capacity of the autonomic and endocrine concomitants of emotion to influence practically every biochemical-physiological process in the body. Elicited adjustive demands will vary as a function of the

perceived strength of threat in the psychosocial situation. The intensity and duration of this threat and the emotional arousal seem to be the relevant parameters determining adjustment (Depue et al. 1979; Goodwin, 1974).

It is to the questions of pathogenesis and recovery, rather than predisposition and initiation, that most research to date has been addressed. Following an outline of depressive symptomatology here, certain main classificatory systems will be presented. Particular attention will be given to the endogenous-reactive debate and to the superordinate unipolar-bipolar distinction of affective illnesses. The concept of depression, as viewed from behaviourist, psychoanalytic, and biochemical viewpoints, will be introduced. Finally, findings from evoked response studies, pertaining to affective disorders in particular, will be presented. Such a broad approach is recommended so as to develop an understanding of depression from a multidimensional viewpoint.

2.1. Symptoms in Depression.

This section will attempt to describe the different clinical pictures that patients can present and to point to areas of controversy. Depressive illness may take different forms and certain symptoms are taken as characteristic of these. Variants, which will be discussed in more detail later, include endogenous, reactive, and anxious depression. However, the existance of such forms is itself controversial and, even for a single variant, agreement is lacking between clinicians as to which symptoms to include and their importance. As Seligman (1974) notes, depression is a convenient diagnostic label which denotes a constellation of symptoms, no one of which is necessary. The relationship between depressive phenomena may be described as a family resemblance, a complicated network of similarities overlapping and criss-crossing. The diagnosis of depression is

thus based on a cluster of symptoms. It is over precisely which phenomena are noteworthy and the nature of the relationship that debate rages. Traditionally, clinician ratings are relatively unreliable and likely to be biased by the preoccupations of the rater. The introduction of standardised interview techniques and operational definitions ofsymptoms have not completely eliminated such unreliability or bias (Kendall 1977).

Depression is defined as "an emotional state with retardation of psychomotor and thought processes, a depressive emotional reaction, feelings of guilt or criticism, and delusions of unworthiness " (Ferster 1974). The label "denotes passive individuals with negative cognitive sets about the effects of their own actions, who become depressed upon the loss of an important source of satisfaction" as well as "agitated patients who readily initiate active responses, and who become depressed with no obvious external cause" (Seligman 1974). It is characterised as a loss of involvement in social life by Matthews and Miller (1972). A unifying theme is added by the prevailing mood of sadness, helplessness and hopelessness (Kendall 1977). The functional symptoms, cited by Ferster (1974), include the major features of reduced frequency of many normal activities. Depression often follows a drastic change in personal circumstances as, for example, in bereavement, loss of health, status, or security. Psychomotor retardation is in evidence, along with lack of interest in previous hobbies and in emotional attachment to other people. The depressed person's perception of himself and others is distorted so as to demonstrate extreme feelings of self-blame, or incompetence, or indifference to, and rejection by, those around him. Prominent bizarre or irrational behaviour which are repetitive and not useful, e.g. hand-wringing, are seen. Frequent weeping and threats of, or attempts at, suicide are common. The depressed person makes frequent complaints or requests for help. Seligman (1974), supporting the behavioural pre-eminance given to passivity, notes also that

current behaviour is of reduced amplitude. Depressed patients are set to interpret their own responses, when they make them, as failures or doomed to failure. For Seligman, the depressive's sense of helplessness, hopelessness, and powerlessness are of central significance. The [illegible] of self-preservation and [illegible] by Beck (1974) who also cites sleep disturbance as [illegible]. He observes that the normal goals of maximizing [illegible] and minimizing pain are inverted.

Fowles and Gersh (1979) list further symptoms said to distinguish endogenous depression. Such patients are seen to unresponsive to external events and were psychotic, whereas reactive or psychogenic depressives were emotionally affected by events. Depressive symptoms are more pronounced in endogenous depression. Rather than indulge in self-pity and blame the environment, as with reactive depressives, endogenous had feelings of guilt, remorse, and unworthiness. Characteristic sleep loss occured in the middle of the night and early morning. Depression was also more marked in the morning. Other cited features include retardation, visceral symptoms, decreased sexual potency and interest, loss of menses, weight loss and general lassitude. These two forms of depression are separable for Kendall (1977) and Matthews and Miller (1972) in that endogenous depression presents a severe unvarying depression, often with acute onset, and accompanied by retardation, guilt, diurnal variation of mood, severe insomnia, and weight loss. Reactive depression consists of a milder illness, often accompanied by anxiety, likely to vary from day to day, with self-pity and histrionic outbursts rather than guilt, and anorexia rather than weight loss. As an illustration of the degree of contention over the status of separable illnesses, the symptom of 'precipitating circumstances' may be considered. Both Kendall (1977) and White (1977) argue that the majority of depressive illnesses are preceded by stressful events of one sort or another, and that truly endogenous illnesses are therefore quite rare. The latter goes further to state

that, with sufficient time and attention devoted to family history, some significant loss may be demonstrated as an aetiological factor.

Gersh and Fowles (1979) argue that anxious depression should be separated from both neurotic and endogenous depression. Quoting Paykel et al. (1971), they argue that a group of anxious depressives can be identified on the basis of high scores on somatic and psychic anxiety, depersonalisation, obsessional symptoms, and fatigue. They are moderately depressed, have high scores on suicidal tendancies, previous episodes of depession, and low scores on reactivity to the environment during the depression.

2.2. Introduction to classificatory schemas.

Kendall (1977) accounts for the number of classificatory schemas as emanating from disputes over the nature and classification of mental illness as a whole, whether mental illnesses are illnesses or reaction types, whether they are independent entities or arbitrary concepts, whether they should be classified on the basis of their symptomology, their aetiology, or their pathogenesis, and whether they should be portrayed by typologies or dimensions. Such diversity is reflected in the lack of commonly acceptable definition of such terms as reactive, neurotic, endogenous, and psychotic. Although the first and second pairs are often taken as internally synonymous, such a convention is not universally adopted (Kendall 1977; Fowles and Gersh 1977). American writers confine the term 'psychotic' to illnesses characterised by delusions, hallucinations and gross lack of insight, the term 'reactive' to illnesses preceded by stressful events, and the term 'endogenous' to illnesses lacking such precursors. Kendall (1977) introduces the nomenclature of Type A and Type B depressions, conforming to his endogenous and reactive types outlined above, and this will be applied in this section.

In addition to the endogenous-reactive and unipolar-bipolar distinctions, discussed below, mention may be made of other typologies and classifications. Overall et al. (1966) on the basis of factor analysis of responses of 160 depressives on their Brief Psyciatric Rating Scale identified three distinct profile clusters. The first and largest group consisted of patients who were anxious as well as depressed and generally presented a clinical diagnosis of neurotic or depressive neurosis. The second group consisted of patients who were notably irritable, and often anxious or agitated as well. The third group, presenting retarded depression, corresponded to the Type A stereotype. (Kendall 1977).

Payek (1971) identified four groups on the basis of cluster analysis. The first and smallest corresponded with the Type A pattern. An anxious group, reviewed above, were elderly and had a history of previous illness. The other groups, hostile depressives and young depressives with personality disorders, were both younger and less severely depressed with marked hostility characterising the former and an underlying personality disorder the latter.

Robins et al. (1972) introduced the distinction between primary and secondary affective disorders. The former are manic or depressive illnesses not preceded chronologically by any other psychiatric illness. Secondary disorders are either preceded by another psychiatric illness, e.g. anxiety neurosis, alcoholism, or accompanied by a life-threatening or incapacitating physical illness.' Primary disorders are further divided into unipolar and bipolar types. Although a primary-secondary distinction has certain advantages in that it can be applied on simple operational criteria, avoids semantic confusion surrounding the endogenous-reactive distinctions, and eliminates all those whose depressive symptoms are implicated in other disorders, little recommends it except as a convenient strategy (Kendall, 1977).

2.2.1. The endogenous-reactive distinction.

The controversy over the nature of the relationship between Type A and Type B depression is cited as the most enduring in modern psychiatry by Kendall (1977), who identified five different formulations of the relationship. These represent an extension of the monist-dualist positions discussed by Fowles and Gersh (1979). Mobius, in 1893 and recorded by Becker (1974), was the first to argue for a differentiation of endogenous from reactive depression based on aetiological distinctions. Emotional reactivity during the illness to the environment was the basis adopted by Kraepelin for distinguishing endogenous from psychogenic depression. He equated reactivity during the depression with a psychogenic, and nonreactivity with an organic, aetiology. This position is currently maintained by Mayer-Gross and Roth in the 'Newcastle school'. Carney et al. (1965) demonstrated a bimodal distribution of scores on functions derived by multiple regression analysis as applied to clinical ratings of 129 depressed patients diagnosed as 'endogenous' or 'reactive'.

By contrast, Mapother (1926) suggested the endogenous-reactive dichotomy was false and that a single depressive illness differing only in severity existed. Although differences may exist between cases of mainly hereditory causation and those in which environmental causes predominate, such clinical differences as precipitating event, emotional reactivity during depression, and outcome failed to distinguish them (Fowles and Gersh, 1979; White 1977). Kendall (1977) also argues for a continuous distribution, but one based on a psychotic-neurotic continuum, as a convenient way of acknowledging the apparent lack of any valid boundary between Type A and Type B illnesses. Response to electroconvulsive therapy and global outcome at ten months have both been shown to correlate highly with a patient's position on the psychotic-neurotic dimension, and in general it appears that this position provides a better index of

outcome than either a Type A or Type B diagnostic label or any individual symptoms. However, as Kendall (1977) himself notes, such an axis does not reflect the diversity of depressive illnesses, nor distinguish between differences in severity or between patients with and without mania. Eysenck (1970) demonstrated that two dimensions are necessary to adaquately represent the variation in symptomology exhibited by depressed patients. The dimensions of psychoticism and neuroticism could describe a patient's position a single continuum.

Since Type B depression is diagnosed in the absence of Type A symptoms rather than by the presence of distinctive pathognomic features, Foulds (1973) pointed out that this constituted a hierachical relationship. He demonstrated that the pattern of symptom ratings derived from self-report questionaire responses of depressed patients fit a hierachical model which discriminated patients with 'Delusions of Contrition' from those without.

Attempts have been made to resolve the question as to whether patients or symptoms can be meaningfully grouped through the application of statistical techniques. A statement of certain considerations, prompted in part by the practitioner's inadaquate understanding of statistical interpretation, seems apposite here. Where the primary data consists of clinicians' ratings, the resulting factors may reflect only the rater's biases and implicit theories, all be they held in common, rather than patient's symptoms. Secondly, wide variation exists in the particular statistical technique applied which, together with the often generated differing results, make comparison impossible. Third, factor analytic techniques require certain assumptions about nature, i.e. symptoms as they naturally occur do group themselves into factors or dimensions. In clinical studies factors represent an intercorrelation between symptoms studied, and do not ipso facto form diagnostic types. Thus, the identification of two or more factors does not allow the creation of diagnostic sub-types to correspond to

such factors. Classification, being concerned with the relationship between individuals, is not permissible on the basis of derived factors; rather cluster analysis on the basis of derived factors may be appropriate (Fowles and Gersh 1979; Kendall 1977).

Mendels and Cochrane (1968) reviewed seven factor analytic studies which had extracted a factor corresponding to endogenous depression. They counted the number of studies in which a given symptom loaded in a positive or negative direction on the endogenous factor. Perfect agreement in all studies was found for the symptoms of retardation, loss of interest in life, visceral symptoms, absence of precipitating stress, middle of the night insomnia, and not showing self-pity. Areas of fair agreement on this factor, in which at least three-quarters of studies agreed, were older age, previous episodes, weight loss, early morning wakening, self-reproach or guilt, suicide attempts, not showing hysterical traits, and not showing an inadaquate personality. Other items, although failing to meet the above criteria, that loaded only on the endogenous factor included delusions, hallucinations, and a previous manic attack. Agitation was not consistently associated with endogenous depression.

Inspection of the Mendels and Cochrane tables reveals that, except for reactivity and the presence of a preceding event, items rarely loaded as highly on the reactive factor as on the endogenous. Consequently, as they also suggest, depression may be involved in a variety of other disorders not meriting the diagnosis 'depression'. Alternatively, as neurotic depression may be diagnosed in the absence of endogenous symptoms, it may consequently result in a more heterogeneous group of patients.

Paykel et al. (1971), based on an assistant's assessment of precipitating events and patients' self-completed Maudsley Personality Questionnaire, supported the association between event initiation and neuroticism with neurotic depression. Pilowsky et al. (1969), employing clustering of

patients' self-ratings, obtained two depressive subtypes; one of which corresponded to endogenous depression and the other being less clearly defined.

In summary, evidence suggests the existence of a fairly well-defined endogenous symptom cluster and a vaguer neurotic symptom cluster (Fowles and Gersh 1979; Kendall 1977). Endogenous depressives lack reactivity to the environment, have fewer precipitants, and do not have hysterical or neurotic traits, whereas neurotic depressives are thought to show the reverse. This distinction is supported by electroconvulsive therapy studies which suggest a more positive patient response for endogenous than for reactive depressives. Also some endogenous depressives show a specific response to treatment by tricyclic anti-depressants, whereas neurotic depressives show a good response irrespective of treatment (Fowles and Gersh 1979). The atypical reactivity of anxious depressives with respect to either precipitants before the onset of illness or during depression suggests that this group should be separated from the conventional neurotic depresssive group (Gersh and Fowles 1979).

2.2.2. The unipolar-bipolar classification.

The separation of patients having alternating manic and depressive episodes from those having recurrent depression alone into bipolar and unipolar groups was first made by Leonard (1957), but it was Angst (1966) and Perris (1966) who first produced evidence for such a distinction. Perris (1966) demonstrated that bipolar illnesses started on average fifteen years earlier than unipolar and recurred more frequently. As attendant personality differences, bipolar were seen as warm, energetic and extraverted, and unipolar as retiring, tense and anxious. This proposal has been reformulated, in part due to its inadaquacy to reflect fully differences in family background, and has been reviewed by Depue and Monroe (1979).

Phenotypic behaviour is invariably combined with the family history of the individual, insofar as it contains the presence, or absence, of manic or hypomanic disorder in first degree relatives, to determine polarity. Inclusion of the patient in one of five bipolar groups is made on the basis of mania, with or without depressive episodes either in the patient himself or in his immediate family. The unipolar group consists of the remaining, recurrent depressions forming a single homogeneous group. It contains the traditional involutional, neurotic and psychotic reactive types where there is no personal or family history of mania or hypomania. It is divided into unipolar 1 and unipolar other on the basis of whether the patient has been hospitalised or treated as an outpatient respectively. The differentiation of the unipolar group, according to the endogenous-reactive and anxiety depression criteria, has been reviewed above. Although bipolar groups are typically hospitalised at an earlier mean age (early thirties) than unipolar depressives (early forties), Perris (1973) suggests that the danger of diagnosing a unipolar disorder for a patient, who later develops bipolar symptoms, can be contained by adopting a defining criterion of three depressive episodes without mania. Diagnosis, with its indicated avenue of treatment, is clearly of some importance.

Although comparisons between these groups have not yet been fully explored, certain findings exist which contribute to our understanding of depression (Depue and Monroe 1979). Psychomotor retardation is exhibited by bipolar patients in the depressed phase, and this is more marked than that of unipolar patients. Bipolars tend to exhibit a sleep disturbance of hypersomnia (>8 hours) and restfulness during sleep, whereas unipolars demonstrate hyposomnia (<6 hours) and who report more trouble falling asleep and more awakening during the night and early morning. Somatic complaints and evidence of anger are more marked in unipolar patients. [illegible] are higher with bipolar disorders, who also demonstrate a worse intramorbid personality. Thus, for

example, divorce rates are higher [illegible] periods of mania. Although the [illegible] of environmental variables has not been studied, [illegible] mode of transmission is implicated for bipolar [illegible] and a polygenic one for unipolar. [illegible] that such findings are tentative.

2.3. The concept of depression.

As a complement to the foregoing clinical and classificatory sections, the following discussion is introduced. Certain behaviourist and psychoanalytic descriptions of depression are presented which, besides adding 'flesh to the clinical bones', will give some hint of the diverse factors identified by different practitioners as important in describing, and accounting for the development of, depression. Finally, the contribution of the biochemical approach will be outlined.

2.3.1. Behaviourist approaches.

Beck's (1974) treatment of depression is a cognitive assessment and interpretation of behavioural data. Of significance is seen to be the depressive's sense of loss, or articulation of personally lacking some essential attribute for success whether it is, for example, social, careerwise, or tangible possessions. The depressive characteristically misinterprets or exaggerates loss, or attaches overgeneralised or extravagent meaning to loss. This theme is the basis for distortion and illogical thinking, which includes selective abstraction, arbitrary inferences and magnification. This notion of loss -past, present, and future- saturates a cognitive triad of the patient's evaluation of himself, his world, and his future. The descriptor 'loser' is apt in that it captures the depressive's appraisal of himself and his experience, and that he is a 'misfit' and inadaquate to meet responsibility or attain goals. Loss itself is idiosyncratically meaningful, and centres on the 'hypothetical loss' or 'pseudo-loss' in that real events, whether positive or neutral, are construed as if negative or disasterous. A motivational deficit, in extreme cases expressed as a 'paralysis of the will' is manifested by the depressive in his antipathy toward activities in which he formerly spontaneously engaged. Any physiological depletion hypothesis is contradicted by the depressive's observation that he feels a strong desire to avoid normal activities. That disturbances in affect and motivation are the outcome of negative conceptualisations is supported by correlational studies between the Beck Depression Inventory and measures of pessimism ($r=0.56$) and negative self-concept ($r=0.70$). Suicide wishes and attempts are regarded as extreme attempts to escape from personal misery and from acting as a burden on family and friends.

The depression-prone person has been selectively sensitised during personal development to certain life events, such as loss of a parent or chronic rejection by peers.

Such traumatic incidents predispose the individual to overact in analagous situations in later life so as to make extreme or absolute judgements. Depression-precipitating circumstances may be specific, e.g. the disruption of an important relationship, loss of job, or may be incidious stresses, e.g. the gradual withdrawal of affection by the spouse. In both types of situation the individual experiences a loss of substantial significance. Such an individual extracts personally relevent, and often simplistic, meanings from these situations. Negative self-descriptions expand to the point of obscuring valued personal attributes and completely colour self-perception. Each new experience is interpreted in terms of this deficiency and, constituting a fresh loss, contributes to the reservoire of accumulated losses. This process of selective abstraction demonstrates the individual's specific sensitivity by which he extracts elements suggestive of loss and glosses over or ignores other features not consonant with this interpretation.

Ferster (1974) presents a behaviourist interpretation in which depression is seen as a loss of behaviour. This can come about in two ways. The frequency of items in a person's repertoire may be reduced to zero by a drastic change in personal circumstances. Alternatively, a person may fail to make the necessary changes in their behavioural repertoire as are required by the processes of growth and development, e.g. physical changes associated with ageing. In this situation, schedules of reinforcement, as determinants of the frequency of a performance, are particularly interestng. Fixed ratio schedules require large ammounts of behaviour to produce a change in the environment and may, speculatively, characterise the situation of the middle-aged.

Behavioural analysis requires examination of how the environment prompts and otherwise controls a person's activities, rather than the way he percieves his own and the outside world. Mentalistic approaches are seen to less useful than a behavioural, which allows the focusing of

attention on how a person comes to act distinctively to the various features of his internal and external environments. An inaccurate observation by him of the surrounding environment introduces the probability that a large proportion of behaviour will be unsuccessful and go unrewarded. The distortion of the depressed person's perceptions reduce the possibility of positive reinforcement because the effect of such distortions is to eliminate performances that cannot be reinforced. Such a failure by the environment could cause or exaccerbate depression. In general, the ability to observe the environment depends on a high enough frequency of interaction with it in terms of successful and unsuccessful reinforcements tailoring performance to the environment in which it can be reinforced.

All the symptomatic behaviours characterising depression are, functionally speaking, avoidance and escape behaviours; technically they are behaviours reinforced by the removal of an aversive stimulus, suicide being the ultimate expression. These behaviours are important insofar as their occurence pre-empt the normal repertoire or are symptomatic of defects elsewhere. Escape and avoidance behaviour, as an internalised passive response to the environment, has two aspects. First, the behaviours emitted tend to be negatively reinforced by aversive stimuli from other people. Second, the passive, depressive repertoire fails to deal with, to avoid, or to escape from aversive social consequences. Reactive depression thus appears functionally defined with the common denominator of decreased frequency of many different kinds of positively reinforced activity. Accordingly, no single cause or psychological process will be responsible because the frequency of items in a depressed person's repertoire is the product of many different psychological processes. The depression is maintained by the depressive's limited, lousy or unchanging view of the world.

Emanating from his studies of induced helplessness with dogs, Seligman (1974) drew parallels between that state and that of depression in humans. Learned helplessness, as

extended to humans, was an attempt to understand depressions in which the individual is slow to initiate responses, believes himself to be helpless and powerless, and has a negative outlook on the future which began as a reaction to having lost control over gratification and the relief of suffering.

In a summary and modification of the theory, Abramson et al. (1978) stated that learning that outcomes were uncontrollable resulted in motivational, cognitive and emotional deficits. The basis of the hypothesis is that the expectation of uncontrlability produces helplessness. Motivational deficits consist of retarded initiation of voluntary responses and is the consequence of the cognitive expectation that events are uncontrollable. The expectation of the ineffectiveness of responses leads to the decreased liklihood of their emission. Further, learning that an outcome is uncontrollable results in a cognitive deficit and interferes with later learning that responding can influence outcomes. Depressed affect itself is not a consequence of learning that outcomes are uncontrollable, but rather that bad changes will occur. The theory was further modified by the incorporation of attribution theory to account for the effects of uncontrollability. The causal attributes that a person makes, as to their helplessness, determines the generality and severity of helplessness deficits and the affect on self-esteem. The relation of locus of control is also of importance. Both personal and universal helplessness situations encourage expectations of noncontingency and consequent future helplessness. The two may be distinguished, however, in that, in the latter, the attribution that no-one can solve the situation is made, and, in the former, subjects believe that they personally cannot solve soluble problems. In this case, self-esteem suffers. Depressives apparently make internal, global and stable attributions for failure and may make external, specific, and less stable attributions for their success.

The authors summarise the model in the following four

postulates. Firstly, depression consists of four classes of deficits: motivational, cognitive, self-esteem, and affective. Secondly, when highly desired outcomes are believed to be improbable or highly aversive outcomes are believed to be probable, and the individual believes that no response in his repertoire will change their likelihood, (helplessness) depression results. Thirdly, the generality of the depressive affect will depend on the generality of the attribution for helplessness, the severity of the depressive defects will depend on the stability of the attribution for helplessness, and whether the self-esteem is lowered will depend on the internality of the attribution for helplessness. Finally, the intensity of the deficit depends on the strength or certainty of the expectation of uncontrollability and, in the case of the affective and self-esteem defects, on the importance of the outcome.

Physiological states, postpartum conditions, hormonal states, loss of interest in reinforcers, chemical depletions and so on may produce depression in the absence of the expectation of uncontrollability. However, there exists a subset of depression -helplessness depression- that is caused by the expectation of response-outcome independence, and displays the symptoms of passivity, negative cognitive set, and depressed affect.

2.3.2. Psychoanalytic perspectives.

Disagreement among psychoanalists regarding the psychopathology of depression has prevented the emergence of a unified, comprehensive, and precise psychoanalytic theory. However, White (1977) identifies certain themes in the literature that are significant.

The affect of depression is a response to the loss (or threat of loss) of someone, something, or some physical or psychological function which is an important source of security and satisfaction. The loss of crucially important

sources of security precipitates a depressive condition characterised by helplessness and hopelessness. The surrender of the ability to cope reduces the person to this state of depression and is one last unconscious cry for help. The very stripping of defenses itself becomes a defensive manoevre. This loss, which generates depression, may be real, fantasised, or symbolic. Most commonly significant depressions involve some combination of all three. The affect of depression derives from the biologically innate response of the infant to the loss of either, or both, of physical and emotional contact with the mother in the critical period from approxiamately six to thirty-six months. Such loss disrupts the normal development of the child and creates a greater than normal vulnerability in later life to the loss of people, capacities, possessions, and circumstances which provide security. The infant, suffering physical and emotional separation, goes through phases of protest, despair and, finally, detachment. It is the effect of separation involving this third phase that is irreversible. The sensitising effect of early parental loss predisposes the person, who has experienced it, to respond to later losses with a greater degree of depression than a person who has not experienced early childhood loss.

Isenberg and Schatzberg (1978) divide psychoanalytic literature into four approaches; these being libidinal, ego psychological, object relational, and cognitive. Representative of this last is the approach of Beck, presented above. Early psychoanalytic theories emphasised libidinal drives and psychosexual development. Abraham postulated orality and ambivalence as key factors in the aetiology of depression. Oral fixation, the result of repeated disappointments in the subject's relationship with the mother, leads to intensely ambivalent adult relationships. When adult love relationships fail anger results, which, when introjected, leads to the lowered self-esteem seen in depressed patients. For Freud, lowered self-esteem provided the key to melancholia. Finding himself in such a situ-

ation, the subject responds with rage to a real or perceived loss and direct the rage first against the object and eventually against the self after the fateful incorporation of the object. Such incorporation, and the antecedent inability to direct anger against the object, is explained by a regression to an oral-narcissistic stage of development. Depressives rely on external sources to maintain their self-esteem.

The growth of interest in ego-functions, their development and their role in psychiatric disorders led to the postulate that depressives responded to their own perceived failure to fulfill their ego ideal aspirations and become depressed as a result of this realization. Such goals, as, for example, to be strong and superior or to be loving, good and non-aggressive or to be appreciated and worthy, all represent aspirations which, in turn, represent a specific libidinal level of development.

Most recently, emphasis has been placed on the nature of an individual's object relationships, their internal representations and their importance to general psychological functioning and psychopathology. Depression, in Kleinian theory, is related to specific developmental tasks which must be resolved in the first year of life in order to obtain any psychic equilibrium. An earlier splitting of an object into 'good' and 'bad' parts must be dealt with by the infant in a defensive position. The infant must deal with the fear of losing the important 'good' object through mastery of the anger generated by the fear. Successful negotiation of this stage is vital to immunity from subsequent serious depressions through the loss of important objects. Winnicott argued that the infant must learn to reconcile the good and bad mother images, but such an attempt produces guilt and tension in the child. This rage must be dealt with in order to develop whole object relationships. In both of these and other formulations, depressive disorders are seen as disturbances secondary to the failure to resolve a basic developmental ambivalence.

In summary, psychoanalytic theory accounts for susceptibility to depression as within the psyche of the individual. This predisposition is rooted in childhood or infancy during which period the mother plays a role of paramount importance. Either her distance or presence, provoking maturational tasks, affects the child's ability in later life to form relationships with other people, set goals, and to act. The fear of loss, guilt, and anger may be re-invoked in threatening situations in later life. Depressive illness, and particularly its expressions of helplessness, hopelessness, and anger, are secondary to the lack of success in overcoming maturational stages.

2.3.3. The biochemical approach.

The biochemical approach to affective disorders has focused on the monamine transmitters dopamine and noradrenaline (catecholamines) and serotonin (indoleamine). Anti-depressant drugs have been observed to interact at various points with amine metabolism; the biogenic amines being among the first substances to be recognised as neurotransmitters. All three amines are present exclusively in neurones in the mammalian nervous system with relatively low concentrations in the cell body and very high concentration in the nerve terminals (Iversen 1973). These neurones have their cell bodies primarily in the upper brain stem and midbrain with projections throughout the limbic system, cerebrocortex, neocortex, hypothalamus, lower brain stem, and spinal cord; these being implicated in the regulation of memory processes and affective states. Noradrenaline may act as a transmitter in the sympathetic nervous system and serotonin in the sympathetic (Wehr-Malherbe 1976; Weir and Goodwin 1977).

Neuronal activation involves a complex chain of synthesis, storage, and release and uptake of neurotransmitter substances which facilitate receptor activation. A defect

in any of these would affect cell function and, whilst pharmaceutical intervention may correct any imbalance and encourage behavioural changes, the precise point of action of any psychotherapeutic agent is impossible to specify. [illegible] is stored in [illegible] from the synaptic cleft. Noradrenaline is considered to two stores in the nerve. The physiological is released from one store (NA1), located close to the [illegible], in response to nerve stimulation via pathway 1 (see Fig. 2).

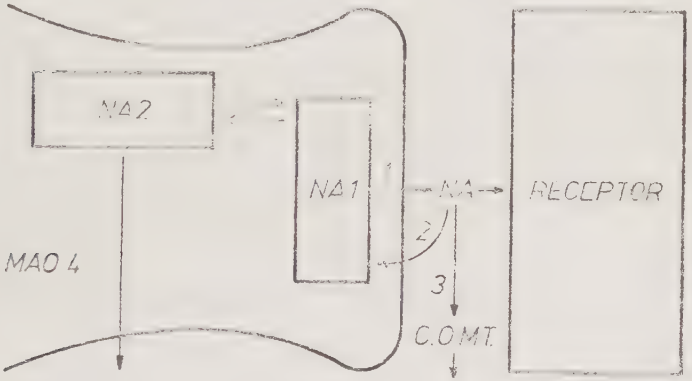

Figure 2. Functional diagram of noradrenaline activation at the nerve-ending (from Granville-Grossman 1971).

After release 95% is re-uptaken for [illegible] (pathway 2) while the remaining is inactivated by catechol-o-methyl transferase (COMT) [illegible] pathway 3. [illegible] is in equilibrium with the first, and its size is regulated by monoamine oxidase (MAO) through pathway 4. During short-lived stress only the first functional store (NA1) is depleted, but with stress of increasing duration both pools are used. Short-term stress affects dopamine in the same way as noradrenaline, but not serotonin, the [illegible] of which is affected by longer term stress. The newly synthesised and retrieved transmitter in store 1 is also used at a

different rate to the former, having a shorter half-life that the newly retrieved noradrenaline.

The catecholamine-indoleamine hypothesis of affective disorders postulates a deficiency in the activity of brain amines in depression and a hyperactivity in mania. Developments, subsequent to his initial formulation of this hypothesis Schildkraut (1965), are reviewed by Schildkraut et al. (1977). Such changes can be absolute or relative. In absolute deficiency or excess, the ammount of transmitter made available in the synaptic cleft is decreased or increased. With relative changes, however, it is rather the number or accessability of receptor sites, the receptor itself, or the activity of synergistic or antagonistic systems that is altered. Changes in the availability of the transmitter may be due to changes in the activity or quantity of synthesising or metabolising enzymes, or in the function of the storage, release, and uptake mechanism. Changes in receptor sensitivity could involve changes in molecular configeration or changes in the function or availability of electrolytes. Behavioural and pharmaceutical evidence is cited for this hypothesis (Granville-Grossman 1971; Wehr-Malherbe 1976).

The complexity of affective illness itself suggests the involvement of widely distributed integrative networks. The correlation of adrenergic systems activity with vigilance, motor activity, and arousal and of adrenergic deficiency with sedation, apathy, and inertia corresponds to the symptomatology of mania, on the one hand, and depression, on the other. Also, a deficiency of serotoninergic activity could be responsible for the depressive aspects of restless- [illegible] (Wehr-Malherbe 1976).

Turning to the pharmaceutical contribution, certain drugs can increase the levels of catecholamines at functionally important receptor sites in the brain. Conversely, drugs known to reduce brain amines, e.g. reserpine and tetrabenazine, lead to sedation and inactivity in animals and can precipitate depression in man (Granville-Grossman

1971; van Praag 1977; Wehr and Goodwin 1977). Anti-depressant drugs, implicated in increasing the availability of amines, are amphetamines, tricyclic anti-depressants of the imipramine-amtyptamine group, and monoamine oxidase inhibitors. The mode of action of these drugs are different. Amphetamines facilitate the release of noradrenaline and block its re-uptake from the synapse. MOA inhibitors prevent the interneuronal deamination of monamines, i.e. inhibit pathway 4, and so increase their concentration in the brain. The greater the anti-MOA activity, the greater its reported anti-depressant effects. The delay in therapeutic response following MOA inhibitor treatment is also found to correspond with the delay in the rise of serotonin (5-HT) levels. MOA inhibitors also block the re-uptake of noradrenaline from the synapse. Tricyclic anti-depressants most probably acheive their effect through blocking the re-uptake of noradrenaline so ensuring higher concentrations for longer periods of time in the synapse (Granville-Grossman 1971).

Monamine treatment effectiveness has been evaluated by reference to, on the one hand, behavioural changes and, on the other, metabolites in cerebrospinal fluid (CSF) and urine. However, caution needs to be exercised in connecting behaviour with underlying biological activity and drug effects. Concerning peripheral measures, questions remain concerning the origin of measured metabolites and their specific relationship to particular illnesses. Probably global, tonic states of amine metabolism or function, including phasic shifts, rather than more rapid, discretely localised changes in brain amine systems, are so accessable (Stinnet and Mendels 1973; Wehr and Goodwin 1977).

Studies of monamine involvement in the pathogenesis of depressive disorders have focused on either their metabolites or precursors. Wehr and Goodwin (1979) state that the available data on catecholamine metabolites in depression do not support a straight-forward biological continuum containing depression as one pole. CSF studies

indicate a dopamine deficiency in only some depressed patients. Similarly, only some researchers have found the catecholamine metabolite to be depleted in CSF and urine. Schildkraut et al. (1977) suggest that abnormalities in secretion reflects distinct diagnostic groups. Thus, excretion rates of this metabolite are lower in bipolar than unipolar depressive types. Excretion rates are also higher during periods of mania and lower during depression.

[illegible] the ideoleamine contribution to the theory, studies of peripheral parameters have failed to produce definite indications of a defect in serotonin metabolism, nor any deficiency in the source substance 'tryptophan'. However, such reports concern overall 5-HT metabolism, which may not apply to the CNS. Indications exist that administration of tryptophan accumulates in the CNS and does not convert to serotonin. A decreased concentration of serotonin metabolite in CSF is fairly regularly reported by some researchers; this being more marked in unipolar than bipolar patients. However, no relationship with severity of depression has been established. Disordered serotonin is also encountered more frequently in 'vital' (endogenous) depression (van Praag 1977). In short, the catecholamine hypothesis, as an explanation of depression, is not adaquate and may better correlate with the level of psychomotor activity (Goodwin 1974).

Burns and Mendel (1977) present a review of catecholamine-indoleamine precursor studies. The majority of the following report is of depressives with no manic symptoms. Two chemicals have been administered in these studies; tryptophan and 5-hydroxytryptophan, which is the [illegible]t serotonin precursor. These will be reviewed in turn, followed by reports relating to the catecholamine pre[illegible]sors.

[illegible]rns reported L-tryptophan as ineffective against [illegible]ression or insomnia, but as an uncontrolled study it is difficult to evaluate. The same criticism applies to a [illegible]f Broadbent, who reported improvement with two-thirds

of patients. Discontinuation of treatment was reported a leading to the reoccurence of depressive mood swings Reports of anti-depressant effect[illegible]ss in [illegible] ECT, have not been supported in replica studies. Several researchers have reported L-tryptophan as effective, o nearly as effective, as tricyclic anti-depressants. However, as they lacked placebo controls, assertions as t their real effectiveness are questionable. It is [illegible] first, that the effectiveness of tricyclics [illegible] e aggerated, and secondly, that non-specific factors, e.g. the treatment setting, affect drug response. Comparison with placebo treatment, either alone or in conjunction wit L-Dopa (a noradrenaline precursor), suggest L-tryptophan tc be ineffective. When administered with a MAO inhibitor, patient response has been shown to be greater than to either drug alone. The effect of tricyclics on depressed mood and anxiety, but not psychomotor activity or arousal, may be potentiated by L-tryptophan, but reports are conflicting.

5-HTP has been studied either alone or in conjunction with a MAO inhibitor or a peripheral decardoxylase inhibitor. Although preliminary results of studies applying 5-HTP alone have used only a few subjects, a remission of depression is suggested. Combination with MOA inhibitor administration provides no convincing evidence for 5-HTP effectiveness. A general problem of interpretation of 5-HTP effectiveness is that it, in large doses, results in serotonin formation in catecholeaminergic as well as serotonergic neurones. Although whole brain serotonin is increased, it is questionable whether 5-HTP serotonin significantly increases in the serotonergic nerves. Thus, serotonin formed after 5-HTP adminisration may not function physiologically. Results from studies administering in conjunction with a decarboxylase inhibitor, which reduces the peripheral conversion of 5-HTP and thus allow proportionately more to pass the blood-brain barrier, suggest an insignificant to moderately favourable response.

Dopamine and noradrenaline levels can be increased by

the administration of L-Dopa. It has been given to depressed patients either alone or in combination with a MOA inhibitor or a decarboxylase inhibitor. Prescribed alone and in small doses, L-Dopa provides no anti-depressant effect. Administered with a decarboxylase inhibitor, it produces either no, or limited, improvement with some retarded depressives. L-Dopa may lead to an increase in activation levels of some patients, but non-responding depressives may become increasingly depressed and paranoid. Doses of more than 4 mg lead to demonstrations of anger.

Since the catecholamine hypothesis of affective disorders suggests that there is a reduction in the functioning of central dopamine and noradrenaline systems in depression, the above results may be taken as contradictory. However, certain qualifications can be made. Firstly, administered doses of L-Dopa were small. Clinical remission cannot depend solely on increases in central dopamine levels consequent on L-Dopa administration. Rat studies indicate an increase in dopamine neurone firing in association with significant increases in striate dopamine levels following administration of L-Dopa in conjunction with a decarboxylase inhibitor. Finally, there is no evidence that L-Dopa produces significant and long-lasting stimulatory effects on noradrenaline levels and synthesis rates.

2.3.4. Evoked-response studies.

Electrocortical investigations present two avenues for research; these being the recording of spontaneous activity or of evoked or event-related potentials (ERPs). In the latter, attention is focused on invariant physiological events occuring between stimulus and response in the nervous system. Whereas the EEG can be recorded directly following amplification, ERPs usually require an averaging proceedure. The sheer volume of data generated, varying in time, space, and voltage, in ERP research needs to be reduced. The first reduction is inherent to the recording itself in that scalp electrodes record activity from diffuse cortical sources; that is the scalp acts as a spatial averager. Computer averaging reduces the obtained signal still further and the selected parameters, describing this signal, are interpreted through the application of statistical procedures. The particular event chosen for investigation offer different advantages (see Section 1). Early evoked potential components might be better for reflecting the more enduring features of psychiatric disorder but they are less influenced by fluctuations in mental state. Any characteristic manifestation of disorder is more likely to influence the later components of the evoked potential or other event-related potentials. Thus, some investigators study early EP components in the hope of finding a central biological defect in schizophrenia or affective disorders, while others look to the late potentials to deliniate states of consciousness of people in altered states of consciousness (Roth 1977). However, as Shagass (1977) notes, it is the double averaged nature of the obtained waveform that renders it highly improbable that they can reflect a detailed pattern of neuronal activity associated with discrete mental events, whatever their degree of normality.

However, certain other considerations can be introduced. Although psychiatric diagnosis presents problems of reliability, they provide convenient reference points ag-

ainst which associated electrophysiological data can be compared. Such an approach is to compare event-related characteristics in defined groups which the expectation that repeatable neurophysiological differences would provide an introduction to defining psychopathological dimensions based on aberrant pathology. Identification of diagnostic differences would permit the determination of that factor in the diagnosis which accounts for the correlation with electrophysiological activity. The lack of concensus on psychiatric classification is, though, often cited as an impediment to such research (Timsit-Berthier et al. 1973; Shagass 1975; Weinberg 1975; McCallum 1979). Vaughan (1975) develops the criticism further. Classification of mental disorder, emphasising qualitative differences in behaviour from normal, impedes attempts to define the nature of brain processes underlying aberrant behaviour. Dignostic categorization results in the emphasis on similarities in symptoms displayed by individuals in the same diagnostic category, and the dismissal of differences or inconsistencies. With the exception of 'organic' disorders, there is no mental illness in which brain processes qualitatively different from those in the normal population have been demonstrated. The unreliability and ambiguity in psychopathological classificatory criteria vitiate any use of such groupings as the basis for seeking fundamentally different mechanisms of disease. Although electrophysiological techniques are indifferent to the nature of the intrinsic and extrinsic sources, including the pathogenic ones that determine them, they can be of great value in quantificative definition of normal and aberrant brain processes. Attempts should rather be directed at defining functional impairments in psychiaric disorders which would allow the application of physiological concepts and methods.

Psychiatric studies of ERPs can adopt one or more different approaches. These being a cross-sectional comparison of clinical populations, longitudinal studies of the same individuals under contrasting phases of the same

illness, or attempts to relate short-term variations in brain potentials to concurrent fluctuations in behaviour. Most studies have taken the first approach (Shagass 1977). Their failure to find distinctive abnormal brain wave patterns 'signiturizing' major functional disorders has led to a re-orientation to an emphasis on quantitative, rather than qualitative, deviations from normal. However, certain points are aposite in the interpretation of results obtained in cross-sectional studies of clinical populations, where these are directed at identifying differences based on basic traits, or on transitory reactions increased in probability of occurence, while the recording is being made. A subject's response to a stimulus, e.g an alerting stimulus, suggest that they reflect state, rather than trait, dispositional factors. However, the relative stability of EEG and ERP measures obtained in longitudinal studies, and evidence of heritability from genetic studies, indicate that trait factors contribute to the variance in brain potentials. To these two contributions must be added that of uncontrollable noise. Comparisons with control populations allow three posibilities. Assuming no genetic contribution to the illness in question and an equal noise contribution, clinical comparison would be on the basis of state factors. Introducing a genetic factor would exaggerate differences and involve the accounting for differences in terms of trait and state contributions. Finally, the state of illness could counteract trait contributions to electrophysiological variance, and again amplify group differences. It is the relative contribution of these sources of variance that makes separation of any groups difficult.

Notwithstanding the above limitations, electrophysiological research offers certain advantages. As it is non-invasive, it offers an ethically acceptable approach to the study of human processes. Secondly, it provides an intermediate level of observation of behaviour and neural activity. Brain potentials can be modified by psychological stimuli and changes in behaviour, and they must reflect

activity in the underlying neural structures. Thirdly, if reliable correlations between brain potentials and behaviour are established, it would indicate that such potentials provide objective indicators of a class of behavioural phenomena (Shagass 1977). Such would also permit a refining of the concept of behaviour that is under study (Callaway 1975). It is further seen to be efficient and objective (Shagass 1972; Walter 1975; Roth 1975). Objectivity is enhanced by the nature of brain potentials in that they are less dependent on previous experience and their measurement requires a minimum of verbal communication (Callaway 1975).

ERPs bear an intimate relationship to psychological state (Roth 1975) and present the major advantage to psychiatry in their relationship to volition and movement, which underlie all behaviour or active mental involvement, and in their exploration of brain functioning at a symbolic level of processing stimuli, making and carrying-out decisions, and reacting to feedback (Dongier et al. 1977). The very slow potentials, e.g. the CNV, reflect events associated with expectancy, motor preparation, interest, and motivation (Shagass 1972). Despite the common acceptance of the applicability of the CNV in psychological research, evaluation of its diagnostic usefulness are very mixed. Whilst Abraham et al. (1976) state that the CNV "has a definite place in the diagnosis of psychiatric disorder" and Shagass (1977a) sees results as encouraging "further application of this new approach to electrophysiological research in psychiatry" opposite opinions can also be cited. "The relationship of CNV amplitude and psychopathology is labile, rather weak statistically, and rarely of help in single cases. The practical application of CNV studies does not seem to have much future in clinical studies" (Dongier et al. 1977). Timsit-Berthier et al. (1973) noted that the study of CNV duration, more than amplitude or morphology, was most revealing and fruitful in the domain of mental pathology. However, as a corollary noted, that the detection of an abnormal CNV wave did not provide a formal

diagnosis, since a non-specific anomaly is found in most clinical categories. Fortunately, the situation concerning the P300 is not as confusing. Although it "bears an intimate relationship of the subject to the environment" (Shagass 1972), it has been poorly, if at all, applied to psychiatric research (Knott and Tecce 1978; Buchsbaum 1979).

In the following sections research pertaining to anxiety and depressive disorders will be presented, together with evaluations of the research methodology applied insofar as it may contribute to clarifying the present state of contradictory statements.

2.3.4.1. CNV studies.

In the following section, research findings will be, to such an extent as is possible, presented according to the research group involved. Attention will be focused first on CNV findings and then, to a lesser extent, on post-imperative negative variation (PINV). Results on test-retest reliability, and intercorrelation between CNV variables, in psychiatric groups will be presented. Particular attention will be given to reactive and psychotic depression. Overviews of clinical CNV studies can be found in Weinberg (1975), Roth (1977), and Shagass (1977b).

In the first report on the application of the CNV to psychiatric populations, Walter (1966) reported that in chronic anxiety and schizophrenic states the CNV developed slowly and irregularly during the signal association and operant response trials, rarely reaching the amplitude of normals. Also reporting a study of 7 neurotic patients, Bostem et al. (1967) found three with no CNV and four with a 'denatured' CNV. Walter (1966), applying an operant conditioning paradigm, suggested that such subjects were not as equally conditionable as normal subjects. With extinction, three or four unreinforced trials, i.e. those lacking the presentation of S2, abolished whatever CNV as had been

developed and it did not invariably recover even after scores of subsequent presentations. No such abolition of the CNV was noted in those suffering obsessional-compulsive neurosis. This difference between high anxiety patients and normal controls is accentuated in distraction conditions where a click was irregularly introduced in the inter-trial interval (Walter 1975). In half of the cases, distraction abolished the CNV (McCallum and Walter 1968; McCallum 1969). Whilst reductions in CNV amplitude (150 msec pre-S2) are also evident in normal subjects, when for example preoccupied with personal worries, psychiatric patients are more prone to such fluctuations (McCallum 1969; Cohen 1974). Whereas the CNV in distraction paradigms with normal subjects reinstated 75% of its original magnitude within 24 trials, high anxiety neurotics and schizophrenics, as a group, re-established only 20-25% and some showed no recovery. Obsessional disorders, with high anxiety as the predominant feature, as opposed to 'constitutional' anxieties, were more similar in the CNV and its recovery during distraction to normals (McCallum 1967). Dongier et al. (1977) report that low amplitude is observed in 14% of hysterical neurotics and almost never in obsessional neurosis. A normal CNV was most usually found with personality disorders. Significant correlations between CNV amplitude in the distraction condition and neuroticism as measured by the Middlesex Hospital Questionaire, are reported; highest correlations being obtained with the factors of anxiety, obsessionalism, and depression (McCallum and Walter 1968). Dongier et al. (1977) speculate that patients with chronic 'characterological' depression have developed homeostatic adjustments to the stresses and or genetic factors, that originate the cerebral dysfunction and clinical syndrome, so that often no electrical sign of the cerebral dysfunction remains apparent.

Timsit-Berthier et al. (1973) produced the first major study of neurotics, with depressive symptoms characterisng the majority, and psychotics, comprised of schizophrenics

and manic-depressives. They reported the following mean amplitudes, of the maximum negativity obtained in the ISI, as controls 16(±6) μv, neurotics 13(±6) μv, and psychotics 11(±6) μv. CNVs of less than 5 μv were found in 8% of controls, 10% of neurotics and in 24% of psychotics. Whilst this did not allow the separation of manic-depressives from schizophrenics, the neurotics' flat, or very low amplitude, CNVs were encountered with 14% of those with hysterical characteristics and never with obsessional. This compliments findings, reported by Timsit et al. (1970), of larger CNV amplitude in obsessionals than hysterics, but which did not allow statistically significant differentiation from schizophrenics. Significantly more hysterics had a high field dependency, as measured on the Rod and Frame test, and had a characteristic (Type A) CNV, in which maximum amplitude is reached early in the ISI. Type B, with slowly rising negativity, is associated with field independency (Pampiglier et al. 1973a; Timsit-Berthier et al, 1973). One criticism can be levelled against this latter study; namely, that the baseline was the visually determined best-fitting straight line 1000 msec prior to S1. Roth (1977) suggests that a reduction in CNV amplitude is more related to anxiety and cognitive disorganisation than specific psychiatric syndromes. He observes, though, that although anxiety neurotics, like schizophrenics, may be more distractable and unable to focus attention, they are often overcooperative and overconcerned about their performance.

Plooij-van Gorsel and Jannssen (1980a) investigated the relationship between extraversion and the CNV in a clinically-unspecified psychiatric population. The effect of white noise on the shape of a biphasic waveform was also studied. Neuroticism scores did not discriminate between high and low extraversion groups. Extraverts produced higher amplitude 'O' and 'E' waves than did introverts, especially in the white noise condition. However, white noise led to a reduction of the 'O' wave in both groups. Amplitude of this component was interpreted as reflecting

the degree to which information is received. Noise was thus seen to produce a reduction in the orienting response (O wave). In a further study, involving neurotic introverted patients and stable extraverted controls in a choice R.T. paradigm, both groups presented higher 'O' waves when the information determining choice was contained in S1 (Blooijvan Gorsel and Jannssen 1980b).

Smaller CNVs are reported in psychotic syndromes, e.g. the depressive delusion syndrome, and in neurotic syndromes, e.g. the worry syndrome (Roth 1975) and in both manic and depressive episodes of affective psychosis (Shagass 1979b). Rizzo et al. (1979) report a study of five cyclothemic patients during different stages of illness in comparison to the normal phase. During the depressed phase a reduction in average area of 24% and in maximum amplitude of 20% were recorded. Reaction time increased from 0.27 to 0.33 sec and response errors from 8% to 15%. In the manic phase, area and amplitude were decreased by 40% and 38% respectively, and R.T. and response errors increased to 0.43 and 24% respectively. No differences between the three stages in CNV latency or latency to peak negativity were observed. This study is, however, questionable in the small sample size, in that EOG artifacts were removed by the use of a potentiometer, and in that no control group was included.

Maximum CNV amplitude of manic-depressives, in either phase, and of schizophrenics are reported as significantly lower than that of controls, but did not discriminate between patient groups. Manics and depressives both showed very low amplitude direct current amplitudes in both response and no response conditions, both between the stimuli and after the response. The finding of no difference between unipolar and bipolar disorder were reported, and that lithium carbonate therapy led to an increase in negativity (Small and Small 1969, 1971; Small et al. 1971). However, these reports are difficult to evaluate due to certain methodological problems. A 0.5 sec ISI was used

which is only marginally longer than the CNV onset latency and so mitigated against the development of maximum amplitudes. Baseline was estimated which could introduce a systematic biasing, bearing in mind Weinberg's (1975) suggestion that depressives have a higher resting negativity in the CNV. Subjects were instructed that their production of artifact damaged trials would prolong the experiment and this would hardly contribute to the rapport advocated by Dongier (1973b) and Dobrovsky and Dongier (1978) as basic to successful CNV recording. Finally, although handidness was not controlled, recording was from one mastoid process.

Abraham et al. (1976) report a controlled study of 42 schizophrenics classified, according to the predominance of florid schizophrenic or manic-depressive features, on the basis of the Present State Examination taken at the time of electrophysiological recording. CNV amplitude (200 msec pre-S2) in acquisition and distraction conditions was lower for schizophrenics, but did not discriminate between manic-depressives and controls in either recording session. With the exception of the distraction condition in the first test, significant differences were obtained between schizophrenics and all others. Severity of illness was associated with amplitude in the distraction condition, and the symptom 'simple depression' among others with the spread of CNV to parietal areas. The authors suggest that the CNV features relate more to the patient's constitution than to his current illness, and that PINV is related to current state. Although this supports differences in CNV amplitude between schizophrenics and normals found by Timsit-Berthier et al. (1973), in their study manic-depressives demonstrated greater similarity to schizophrenics. In a pilot investigation of the effects of ECT, Elton (1974) found CNV area and peak amplitude to be less, and onset latency later, in endogenous depressives in comparison to normals before the onset of treatment. Walter (1975) reports one case of chronic intractable depression with no CNV which, after microcoagulation, demonstrated CNV in the frontal cortex.

Giedke and Selz (1980) compared responses of primary depressives to normals in conditions in which a motor response could not, could, or could under a speed of response criterion terminate an aversive S2. ISI was two seconds. Although R.T. was longer and CNV amplitude lower in depressives than controls, these differences were not significant except for R.T. in the condition of constrained controllability. Both patients and controls demonstrated decreased R.T. and increased CNV amplitudes in the condition of total control of S2, and similar changes with the introduction of a time constraint on the MR. In a further paper, Giedke et al. (1980) report an investigation of 18 primary depressives (15 unipolar and 3 bipolar) under response and no response conditions (ISI 2 sec), with S1 and S2 being identical. Inspection of their tables in these two papers [illegible] lower CNV amplitude and longer R.T. in the simple condition as against an aversive S2. No such differences are reported in the second study between depressives and controls in [illegible] CNV amplitude in the no response condition. In the response condition, controls increased CNV area, but patients did not. No differences between groups in CNV latency were observed. Reaction time in patients was some 66% slower than controls, but at no time did it correlate with CNV area. A high depression score, on the Hamilton Depression Rating, was significantly correlated with a small CNV. Those not receiving medication corresponded to the highest depression group. Thus, those receiving medication had low Hamilton scores and higher CNVs. The authors conclude that CNV reduction seems to be a constant sign of depressive illness. However, Straumanis et al. (1980) failed to demonstrate differences between patients of various diagnoses and normals and, although CNV was enhanced in a condition of response over no response, no differences between strong and weak shock conditions were evident. Paty et al. (1980) report a positive CNV in a case study of 'melancholia'.

Abnormalities in the PINV are more consistently re-

ported. In obsessional-compulsive neurosis, irrespective of the presence of anxiety, negativity, after the operant response, is demonstrated (Walter 1966). Timsit-Berthier et al. (1973) reported that the majority of depressive neurotics had a 'Type 1' PINV (negativity returning to baseline with S2) and that psychotics, including manic-depressives had 'Type 4' (negativity increasing after S2 so as to present a dome shape) or 'Type 3' (with post-S2 resolution delayed, but no further increase). CNV prolongation post-S2 in schizophrenics is elsewhere reported (Abraham et al. 1976), but was not consistently observed in weekly examinations (Knott et al. 1976). Rizzo et al. (1979) observed PINV in four of five cyclothemic patients, but only in the manic phase. Giedke et al. (1980) found no differences in primary depressives, but uncontrollability of an aversive stimulus did produce significantly larger PINV (Giedke and Bolz 1980). PINV is observed in 9-16% of normals, 37-39% of neurotics, 75-91% of psychotics, and in 94% of early schizophrenics (Dongier 1973; Dongier et al. 1974, 1977; Dubrovsky and Dongier 1976; Timsit et al. 1970). A normal PINV correlates with the existence of guilt and anxiety on the BPRS (Dongier et al. 1976) and schizophrenics with normal PINV score higher on the subscales for depression, anxiety, and guilt feelings than schizophrenics with abnormal PINVs (Dongier et al. 1977). PINV and total pathology score on the BPRS and Clinical Global Impression Scale correlate positively. A differential diagnosis between endogenous depression and characterological depression, on the basis of abnormally prolonged PINV with a dome-shaped appearance, is possible (Dongier et al. 1977), and such prolongation is strongly associated with prognostic assessment at discharge (Dubrovsky and Dongier 1976). No reliable differences between manic-depressives and schizophrenics are also reported (Dongier 1973, Abraham et al. 1976).

Turning to stability of CNV measures over time in psychiatric patients, McCallum (1969) reported that CNV amplitude increased in a group of anxiety neurotics retested

after 1-2 months in both CNV aquisition and distraction conditions, but this just failed to reach significance. Similarly, Knott et al. (1976) monitored peak CNV weekly in schizophrenic and depressed patients, and found that negativity increased during hospitalisation, especially in the difficult condition of a discrimination reaction-time task when errors were punished. Psychotics are seen to have less stable CNVs, including the post-imperative part, both within and between sessions (Timsit-Berthier et al. 1976). In a further report (Timsit-Berthier et al. 1978) reliability was investigated in stable patients over periods of 1-2 months and 1-2 years and in unstable patients, mostly improving, over 1-2 years. Both groups of patients suffered psychotic disorder and all received psychotropic medication. Pre-S2 CNV amplitudes were significantly correlated over 1-2 months in the stable group and over 1-2 years in the unstable. Unstable patients demonstrated a significant inverse relationship to resolution mode, changing from resolution types 3 or 4 to Type 1 (see above). Stable patients demonstrated significant correlations over both test periods, predominantly retaining Types 3 and 4 classification of resolution mode. Test-retest reliability for the stable group over 1-2 years was significant for duration of resolution. Abraham et al. (1977) found PINV abnormalities consistent, for schizophrenic and manic-depressive groups, between recording sessions. No changes in amplitude with schizophrenics over a ten week period were recorded (Abraham and McCallum 1977). Straumanis et al (1969), in a group of psychiatric patients, were not able to support the idea that CNV responsiveness was a relatively stable individual characteristic. Little correlation was seen between the changes observed in the same subject on the same day under two experimental conditions.

Verhey et al. (1980) report an investigation of the intercorrelation between CNV measures in two groups of psychiatric patients, of undisclosed diagnosis, with different ISIs. CNV amplitude (100 msec pre-S2) and PINV,

corrected and uncorrected for reaction time, were compared using three different ways of determining baseline: these being 1 sec or 0.5 sec pre-S1, and 350 msec post-S1. Taking a 1 or 0.5 sec pre-S1 baseline did not affect mean CNV amplitude or standard deviation. A negative correlation between pre-S2 amplitude and reaction time in both groups with each baseline was observed. Both CNV amplitude and standard deviation correlated with PINV amplitude, both corrected and uncorrected for reaction time, in the three alternate methods of determining the baseline.

Just as there is a lack of consensus in CNV studies over the interpreted psychological significance of CNV and PINV phenomena, so also are there differences obtained with similar clinical groups. Perhaps not suprisingly then is the applicability of CNV research questioned. In part, however, proper evaluation is made difficult by methodological failings, such as too short an ISI, inadaquate definition of baseline, lack of homogeneity or definition of the psychiatric groups studied, absence of a control sample, and too small a sample size. Differences in experimental procedures are currently being faced in an attempt to standardise recording and diagnostic procedures (Abraham et al. 1980). Further, although the averaging procedure assumes that the evoked responses are time-locked to the evoking stimuli, such ERPs do show some variability. Consequently the failure to report standard deviation in each recording, particularly for psychiatric patients, is suprising. Finally, there is a conspicuous lack of long ISI experiments to separate CNV components and more directly to relate any abnormality in these to psychological deficits in defined psychiatric groups. That the CNV components are reported to reflect different psychological processes makes them particularly suitable for application to assess cognitive, emotional or motivational deficits. Insofar as the early component of the CNV is said to constitute an index of the orienting response, so the degree to which stimuli are received, i.e. environmental reactivity, may be assessable.

Insofar as the later component reflects such processes as expectation or motivation, so may such behavioural deficits as, for example psychomotor retardation, be examined.

2.3.4.2. P300 studies.

In the pre-conference discussion on psychopathology of the 4th International Congress on Event-related Slow Potentials of the brain (see Knott and Tecce 1978), Callaway suggested that later variability in components of averaged evoked responses not having been "tweaked up"; this leading to highly variable later responses which also reflect other things happening in the brain. A differential physiological refractory period is suggested Shagass and his co-workers and by Floris et al. (1968). Varying very short ISIs to investigate recovery cycles, Shagass and Straumanis (1969) found full recovery of S2 responses at about 100 msec and 20 msec. Psychiatric patients were found to have less recovery, i.e. smaller S2 responses in relation to S1, with ISIs of about 20 msec. This delay in amplitude recovery persists even when age, sex, and S1 differences are all controlled. Shagass suggested that this reflected a non-specific health-sickness continuum. This was the conclusion also of Floris et al. (1968) who studied the recovery of the P60-N80-P125 component in psychiatric patients by varying ISI from 100 to 180 msec. They too found that recovery cycles distinguished patients from normals, but not between patients. Attenuation of the major component 400 msec post-stimulus were found by Lelord (see Knott and Tecce 1978) to visual and auditory stimuli in schizophrenics in comparison to neurotics and normals. Giedke et al.(1980) report that the N1-P2 components to S1, in a CNV paradigm, was 25% smaller in primary depressives than in normals in both response and no response conditions. Correcting the data by use of a Woody filter, which corrects for variable latency, showed that depressives' single EPs were significantly less time-

locked than normals. However, this greater temporal variability contributed only negligibly to observed group differences in AEP. Higher rates of increase in middle latency AER amplitudes with increases in stimulus intensity (augmentation) is reported in patients with bipolar affective illness than in those with unipolar (Buchsbaum et al. 1971). The augmentation-reduction hypothesis suggests that individual differences in EP amplitude-intensity relationships may reflect differences in the optimal level of sensory input. However, a search of the literature reveals few reports dealing with the late positive component in clinical populations.

Levit et al. (1973) investigated N1-P3 amplitudes, and standard deviation to these components, in schizophrenics, psychotic depressives, and normals in conditions of certainty and uncertainty concerning the modality of a coming stimulus. Combining data from all conditions, N1-P3 was largest for normals (12.9 μv), medium for depressives (12.2μv), and smallest for psychotics (8.8μv). Whilst differences between schizophrenics and normals and depressives were both significant, there were no differences between depressives and normals. Significantly larger N1-P3 amplitudes were obtained in the uncertain condition than in the certain condition for all subjects, as well as each group separately. Amplitudes in the uncertain condition produced significant differences between both schizophrenics and depressives and normals, but not between the former two groups. Correct guessing of the modality of the next stimulus in the uncertain condition, as opposed to incorrect guesses, although significantly larger for all subjects taken together, was significant only for normals as a distinct group. Similarly, the mean cross-modal N1-P3 amplitude was significantly larger for all subjects taken together than ipsi-modal, but again only for normals as a group. Differences in means of ipsi-, and cross-modal conditions were not significant for either patient group. Significant differences in this measure were obtained be-

tween schizophrenics and both controls and depressives, but not between the latter two. Nonsignificant overall differences in P3 latency were obtained; depressives having the longest and normals the shortest. Significant changes were only obtained for latency in the uncertain condition; namely, increased latency for schizophrenics, and decreased for depressives and normals. All these findings for the N1-P3 component were also found for the P3 alone, i.e. the N1 component contributed little, if anything, to N1-P3 differences between groups. Medication was not found to dramatically alter the evoked potentials. Schizophrenics demonstrated greater variability in P3 than either normals or depressives. A further report on this data (Tueting and Levit 1979) investigated variability in the evoked response measures. The recording session was divided into four segments. Clearest progressive changes were observed in the N1 component, but not necessarily accompanied by changes in the other components. Schizophrenics demonstrated a decrease in P300 to visual stimuli in the certain condition, but the depressive group did not. Normals, as a group in the uncertain condition, demonstrated stability in amplitude and latency, whilst varying individual in an idiosyncratic direction. This is in accord with Sutton's (1977) finding that P300 demonstrates no systematic trends to vary with normals. In the certain condition in response to light stimulation, there was a tendancy for P300 amplitude to decrease; this being most marked for schizophrenics and least for depressives. Intriguingly, schizophrenics demonstrated a concomitant tendancy for P300 latency to increase in this condition, which may have been due to a failure in habituation of the orienting response.

Timsit-Berthier and Gerono (reported in Roth 1977) found reduced late positive wave amplitude in psychotics to infrequent stimuli as compared to normals. Schizophrenics were also found to demonstrate lower P300 than normals to infrequent stimuli in a study by Roth and Cannon (1972); normals also demonstrating a reduction in P300 over a ten

minute period. The short (220 msec) latency, obtained in this study, and the habituation of response in normals may have been brought about by their use of a constant 1 sec ISI. Although not reaching statistical significance, P300 to auditory and visual stimuli in psychotic depressives were attenuated in comparison to normals and reactive depressives (Shagass et al. 1978). The authors suggested that augmentation of N100 and P200 components reflect stimulus set attention and the P300 response set; corresponding to the division proposed by Broadbent (1971). Reduced amplitude in these components could result from impaired attention, possibly resulting from the emotionally disturbed condition of psychiatric patients. However, no discriminating differences were observed between neurotic depressives and controls, and that between psychotic depressives and controls they ascribed to cognitive alterations associated with impaired reality testing (Shagass et al. 1978).

In a study of 12 neurotic introverted psychiatric patients of undisclosed diagnosis and 12 controls in a choice reaction-time paradigm, Plooij-van Gorsel and Jannssen (1980) varied the information guiding choice between S1 and S2 and compared these responses to those obtained in a simple reaction time task. P300 showed a minimal condition effect. Latency for the patient group was shorter than that for normals and tended to increase from the simple reaction time situation to the situation where information was contained in S2; both results just failing to reach significance.

Summarising findings relating to psychotic depressives and normals; the differentiation in P300 found by Shagass et al. (1978) was only evident in the condition of uncertainty in the study of Levit et al. (1973). The absence of amplitude reduction in the certain condition for depressives (Tueting and Levit 1979) suggests that stimuli were equally effective, as far as electrocortical manifestations are concerned, during the recording session. Such an interpretation, emphahsising reaction to the environment, is

supported by elevated amplitudes in the uncertain condition. Overall, depressives demonstrated the longest P300 latency, but curiously this reduced in conditions of uncertainty. This suggests support for the concept of psychomotor retardation in depression. The findings of Shagass et al. (1978) concerning the similarity of reactive depressives and normals may be due to his small group size and to his not manipulating the information content of stimuli. The suggestion of homeostatic adjustment (Dongier et al. 1977), to account for a normal CNV in characterological depression, may be of relevence here. Clearly, further studies are required of P300 amplitude and latency are required in conditions in which the information content of stimuli is manipulated.

2.4. Summary.

In this overview of depression, lack of agreement between clinicians as to the principle symptoms and their developmental basis is apparent. Disagreement extends to the validity of identifying distinct disease groups. Commonly, the complexity of depression, as involving cognitive, motivational, and affective psychological processes, is recognised in how the patient behaves, or fails to behave. Such malfunctioning is rooted in earlier experience and is precipitated by environmental circumstances, with or without any underlying biological disposition. Generally noted, and of interest in the present study, are symptoms involving pessimism concerning personal past, present and future, the intensive and extensive reduction of activity, such that even activities in the reduced range of behaviours are not as purposively pursued as previously, and motivational deficits. Endogenous depression may be distinguished from reactive principally by environmental reactivity. Attempts to demonstrate the validity of these illnesses and their symptoms by external measures, as obtained for example in

biochemistry, have largely failed. Such may be accounted for by the existant diagnostic imprecisions and by the amorphous involvement of neurotransmitters themselves, however altered by medication, in the regulation of behaviour. Nonetheless, the need remains to identify cortical correlates of behavioural abnormalities. The contribution of neuropsychological studies of the CNV and P300 are inconclusive. Even where methodological criticisms do not apply, reigns a lack of agreement concerning the affective disorders. Research efforts, aimed at the reactive depressive variant of these, are conspicuously lacking and attempts have not be made to apply more recent developments in the evaluation of ERPs, in terms of possible psychological prosesses involved, to psychiatric studies.

3.1. Subjects.

Event-related electrocortical activity was subjected to statistical analysis from 19 control normal subjects (Males: 7, Mean age: 26, Range: 18-33; Females: 12, Mean age: 27, Range: 18-40) and 21 reactive depressives (Males: 9, Mean age: 34, Range: 23-53; Females: 12, Mean age: 34.4, Range: 21-41). A smaller group of seven endogenous depressives were also recorded (Mean age: 33.3, Range: 24-41). A further six patients and seven controls were discarded due to insufficient data obtained after trial selection for averaging on the basis of absence of contamination. The psychiatric diagnosis was made by hospital staff; the criteria for a diagnosis of 'reactive depressive' being described by de Jong et al (1981). Reactive depressives were recorded on admission to, and discharge from, hospital, and normals over comparable intervals. Endogenous depressives were recorded on only one occassion as their treatment was too varied to permit further comparisons, and included, in some cases, medication.

3.2. Apparatus.

Subjects were seated in a fully supporting chair in the centre of a dimly-lit, sound-damped room (2x5 m) in a partly reclining position. Red and green lamps (arrow-shaped, 1.75x1.5 cms) were located in the centre of a white screen (1.5x1.5 m), approxiamately at eye-level, at a distance of 3 m. These served the dual purpose of an ocular fixation point, being illuminated 2-4 sec before S1, and for feedback. Warning stimulus (500 Hz, 80 dB, 500 msec) and imperative stimulus (800 Hz, 80 dB, 500 msec), delivered over floor-based loudspeakers (Canton), were generated by separate frequency generators (Wavetec). Stimulus delivery,

feedback, and data collection were controlled on-line by a PDP 8E computer.

Electrocortical activity was recorded from the vertex to left mastoid, applying 9 mm silver silver-chloride electrodes (SLE) with collodion, and the EOG with Beckman electrodes. Electrodes were freshly chlorided for each recording session. EOG was recorded from electrodes located above the interior and below the exterior canthus to facilitate monitoring of both lateral and vertical eye-movement. The patient ground was located in the centre of the forehead. EEG recordings were made at less than 5 kOhms impedence. Amplification settings, on the Beckman Dynograph R, were set at 0.5μv for the EOG and and 0.2 for the EEG. Both were recorded using Beckman Type 9806A A-C couplers set for a 30 sec time constant and 30 Hz high frequency filters. Input to the PDP 8E was limited to two channels.

3.3. Experimental design and procedure.

Subjects were recorded in two fixed foreperiod (6 sec) reaction time conditions of 40 trials each. In the first condition feedback was given in the form of a red light for a 'slow' reaction and a green light for a 'fast' one. The reaction time criterion was manipulated by the experimenter to allow each subject 'wins' on about two-thirds of trials. The second condition was identical to the first except that 25 Pfennigs could be won for each fast reaction. Controls were in addition paid 10 Marks for attendance. The inter-trial interval varied randomly between 20 and 30 secs.

After a variable interval of 2-4 sec, following the onset of the fixation lights, the warning and imperative stimuli were delivered. Subjects were instructed to press a key-switch located on the right arm of the chair in a position convenient to the particular subject. Speed of response was encouraged, but stimulus anticipation discouraged. Subjects were given training, at the begining of

each session, in the control of head and eye movement, which also permitted familiarisation with the experimental procedure. A 5 min pause was taken between conditions to allow subjects recovery and the resetting of equipment.

Subjects were assessed for psychopathy (Beck Scale), present mood (Befindlichkeitskala), handedness (translated from Oldfield 1971) and, post-session, performance self-evaluation. These forms are contained in Appendix A.

3.4. Data analysis.

Each trial consisted of 900 data points over an 18 sec interval, i.e. one per 50 msec. Data was averaged off-line following exclusion of trials contaminated by eye-drift over 20 μv or by eye-blink. Results could be output as single trials, or averaged and output in either digital or graphical format (Interactive Digital Plotter), with or without standard deviation. Averages were referenced to a one sec pre-stimulus baseline on-line by the averaging programme. Full programme descriptions can be found in Strasberger (1980).

Five data points were collapsed into one in the CNV averages for curve smoothing purposes. To exclude distortion of the CNV measures from responses to S1, quantified EEG analysis was delayed 800 msec post tone onset. The CNV measures, automatically displayed, consisted of the maximum amplitude 0.8-2.8 sec and 4.0-6.0 sec post-S1 onset together with their latencies, the corresponding areas, and total negativity over the averaged interval. Standard deviation of the early, middle and late segments of the ISI activity were also determined by the computer.

Two data points were condensed in the P300 averages following S1 delivery delivery and the onset of the fixation lights. The invariant randomising procedure across trials of the PDP 8E permitted an accurate localisation of elicited activity in the EEG record consequent upon light onset. The

major positive deflection following stimulus onset (both tone and light) and the complimentary latency were determined by the programme. Amplitude of this peak to the preceding and succeding negative peaks were also determined, in addition to a peak-to-baseline measure. The standard deviation of the major positive component was also computed.

HYPOTHESES.

Although recent developments have called into question established findings for the CNV, certain aspects of this electrocortical event and the P300 suggest them to be particularly well suited for application to depressive illness. Both provide an index of the degree of involvement in an experimental task as the, albeit artificial, immediate environment. Monetary incentives have been found to produce higher amplitude CNVs in short ISI paradigms, but the question remains as to whether this effect would be manifested in the biphasic waveform, the reliability of which itself is unconfirmed. The development of any second component in the CNV apparently depends on the subject's ability to predict the timing of S2 occurence.

The P300 presents two aspects of interest. Firstly, its invocation indicates the occurence of subjectively important events. Even predictable events can produce P300 when they fulfill this criterion. Secondly, P300 latency is taken as a measure of stimulus evaluation time, but the question remains as to whether this latency can be reduced by important, e.g. with monetary incentives attached, events when decision-making complexity is held constant.

Certain clinical features of depression need to be mentioned in this context, although it is accepted that their importance is open to question. The application of the CNV to behavioural tasks suggests it as particularly suitable to the examination of psychomotor retardation, which is demonstrated in a reduced extent and intensity of activity. Motivation deficits may similarly be reflected in modifications in the later component of the waveform. Psychogenic depression, in contrast to organic, presents reactivity to environmental events. That the early component of the CNV is reported to be related to orienting and the P300 to complex psychological responses to environmental stimulation permits their application to this issue.

Whilst the present investigation is basically exploratory, the above considerations suggest the following hypotheses.

1. In any biphasic waveform in the CNV obtained from normal subjects, the later component may be expected to demonstrate lower retest reliability than the earlier reflecting greater inter-subject variability in task engagement.
2. Depressives, on admission to hospital, demonstrate lower CNV amplitudes and later peak latencies than normal controls in conditions which include and exclude a monetary win incentive. Endogenous depressives demonstrate such differences to a more marked degree.
3. Depressives, on discharge from hospital, demonstrate CNV responses inseparable from those of normal controls.
4. Depressives, on admission to hospital, demonstrate lowered amplitude, and increased peak latency, of P300 in comparison to normals. Such differences are more marked in endogenous depression.
5. On discharge from hospital, reactive depressives demonstrate P300 amplitude and latency indistinguishable from those of normals.
6. More significant events, e.g. those including a monetary incentive, are manifested in the P300 in reduced latency and increased amplitude in normals.

RESULTS.

The results will be introduced so as to answer the previously presented hypotheses. Univariate parametric and nonparametric analyses will be presented first, and the section will conclude with a demonstration of the multivariate approach to the solution. The former procedures are contained in Version 8 of the Statistical package for the Social Sciences (Nie et al 1975) and implemented on the CDC 175 at the Leibnitz Rechenzentrum in Munich. Unless otherwise stated, a significance level of 5% was accepted throughout. Where appropriate, results pertaining to endogenous depressives will be introduced alongside the main presentation of reactive depressives and the control subjects.

Before turning to the statistical analysis, certain observations, based on visual analysis of the grand average waveforms for each group in each condition within each session, are offered. Whilst the inherent dangers in 'eye-balling' the data are recognised, such does, nonetheless, provide a valuable impression of the variables, describing the waveform, as a whole. Appendix D contains grand averages for the CNV and those for the ERP following light and tone onset in Appendices E and F respectively. Means and standard deviations to these variables are contained in Appendix C. The legend to the variable names, found in all graphs and tables, are to found in Appendix B.

5.1. Visual description of the waveforms.

In the folowing presentation, inspection of the averaged EOG discounts without exception, eye-movement as a source of artifact. Except for normals in the first condition, and for reactive depressives with incentives, in session one, biphasicity in the CNV can be seen in all grand averaged waveforms (Appendices D:1 and 2). Whilst normals

demonstrate instability in both early and late peak negativity between sessions in both conditions, reactive depressives demonstrate only an increase in late negativity across sessions in both conditions (Appendix D:3 and 4). Visual analysis of the waveforms in the second session, for reactive depressives in contrast to normals, does not allow any differentiation between conditions (Appendix D:2). By contrast to both of these groups, endogenous depressives do not demonstrate biphasicity; although in the incentive condition an initially depressed early amplitude increases gradually during the ISI to an amplitude marginally elevated over the non-incentive condition. The marked increase in late amplitude for reactive depressives in both conditions of the second session is of interest. However, the relative undifferentiation of waveforms between conditions within sessions, as also for controls in session one, suggests that the introduction of incentives had little effect.

Inspection of the waveforms emitted to light onset for both controls and reactive depressives demonstrated a major positive component (latency: approximately 350 msec) followed by a sustained elevation in baseline negativity (Appendix E:1-4). This positivity appears reliably in averages for both groups and appears of slightly reduced amplitude in the incentive condition in comparison to the non-incentive. Although endogenous depressives (Appendix E:5) also demonstrate this activity the positivity appears overlaid by intrinsic cortical activity, and truncated in the incenive condition. The former artifact may be due to the small number of subjects; and the latter feature suggests a greater temporal variability so that a 'clean' peak is eliminated through the averaging process: the eye-movement apparent in this average may also be responsible. However, the question arises as to what, if any, extent the sustained negativity, following the positivity, may have introduced bias into the measures. Such a consideration has two aspects. Firstly, insofar as absolute CNV measures may be reduced, and secondly, whether such a

baseline deviation distorts group differences over sessions or conditions, i.e. is more evident in one group than the other. The first aspect is more difficult to resolve, but would be important only if some physiological ceiling to negativity were reached. Because tone onset occured at no fixed time-point, i.e. followed light onset by 2-4 sec, it is not possible to determine accurately pre-tone baseline relative to that pre-light onset. Further, since the trials rejected, due to eye-movement, in the ERP and CNV averages were not identical, an exact comparison is not possible. Inspection of the waveforms suggests that, at a point 2 sec following light onset, and constituting the earliest possible tone onset, negativity was minimal, i.e. less than 3 μV, in any waveform. Inspection of the mean standard deviation curves suggests no systematic trend for negativity to increase. This is further reflected in the 1 sec baseline prior to tone onset (see Appendix F) which also demonstrate no trend to an anticipatory increase in negativity. This suggests a non-differential effect between groups or sessions or conditions. It may thus be, tentatively, concluded that any pre-CNV negativity exerted, at most, minimal distortion on absolute amplitude measures and one that, in all probability, would not cause any physiological ceiling to be reached. The above suggests, further, that any distortion was non-systematic, i.e. did not distort session or condition effects or group differences. An exception to the above considerations may be the endogenous depressives in the incentive condition in which baseline, following the positivity, reassumes the pre-stimulation level; eye-movement is not considered to be possibly responsible.

Turning, finally, to the visual evaluation of the ERP to tone onset for the different conditions and sessions, all waveforms demonstrate a marked negative peak (latency: approxiamately 120 msec) and a positive peak (latency in normals: approxiamately 240 msec)(see Appendix F). This positive-negative complex is of reduced amplitude for each

group in both sessions in the incentive condition in comparison to that without. The question arises as to which part of the conventionally-accepted waveform this component might represent, i.e. a late P200, or an early P300, or an amalgum of the two. Whilst this issue will be returned to in the discussion section, certain points can be made here. The succeding waveform (termed N2) demonstrates a slower increase in negativity than the preceding limb of the wave with an occasional notch in the waveform, evident for both reactive depressives and controls, with a latency around 300 msec. However, caution needs to be exercised in transfering waveform analysis between experimental paradigms. A fuller discussion requires the statistical evaluation of the stability of this major component over time, and its responsiveness to experimental manipulation. However, so as not to prejudice later discussion this component will be termed "P" in the subsequent presentation of results. One further feature of interest in the emitted activity following the tone, of latency approxiamately 700 msec, riding on the slow activity evident in all waveforms. Its latency, of some 200 msec after tone offset, suggests this to be the 'off-potential'.

5.2. Test-retest reliabilities.

Pearson correlation analysis was applied to the CNV and ERP measures between and within sessions for both groups separately. Results will be presented in this respective order. Reference to Tables 1a), b), and c) provides full inter-session correlations, and Tables 2a) and b) intra-session.

A highly significant association for controls between the two recording sessions was found only for early CNV amplitude ($r=0.539$, $p>0.009$) in the non-incentive condition. Although not nearing notable significance levels, amplitude, latency and area for the later part of the waveform suggests

VARIABLE	CONTROL	R.DEP.	VARIABLE	CONTROL	R.DEP.
ampl11	0.539	0.010	eegl11	0.770	0.727
ampl21	(0.009)	(0.48)	eegl21	(0.001)	(0.001)
amp211	0.007	-0.128	eeg211	0.618	-0.142
amp221	(0.49)	(0.29)	eeg221	(0.002)	(0.28)
ampl12	0.289	0.357	eeg311	0.502	0.244
ampl22	(0.11)	(0.06)	eeg331	(0.01)	(0.16)
amp212	-0.107	0.469	eegl12	0.109	-0.134
amp222	(0.33)	(0.02)	eegl22	(0.33)	(0.29)
latl11	0.091	-0.033	eeg212	-0.067	-0.201
latl21	(0.36)	(0.44)	eeg222	(0.39)	(0.20)
lat211	0.064	0.566	eeg312	-0.180	0.050
lat221	(0.40)	(0.005)	eeg332	(0.23)	(0.42)
latl12	0.140	-0.119			
latl22	(0.28)	(0.31)			
lat212	-0.067	0.153			
lat222	(0.39)	(0.26)			
arl11	0.206	-0.036			
arl21	(0.20)	(0.44)			
ar211	-0.017	0.205			
ar221	(0.48)	(0.19)			
arl12	0.211	0.205			
arl22	(0.19)	(0.19)			
ar212	-0.202	0.510			
ar222	(0.20)	(0.01)			
tal1	0.008	0.208			
ta21	(0.49)	(0.19)			
tal2	-0.019	0.024			
ta22	(0.47)	(0.46)			

Table 1a): showing inter-session Pearson correlations, and the exact probability in parentheses, of CNV variables for both groups in both conditions.

B

VARIABLE	CONTROL	R.DEP.
lanp11	0.805	0.513
lanp31	(0.001)	(0.02)
lanp12	0.562	0.533
lanp22	(0.009)	(0.01)
lnp111	0.779	0.484
lnp121	(0.001)	(0.03)
lnp211	0.766	0.468
lnp221	(0.001)	(0.03)
lnp112	0.533	0.523
lnp122	(0.01)	(0.01)
lnp212	0.539	0.486
lnp222	(0.01)	(0.02)
llat11	0.421	0.161
llat21	(0.05)	(0.28)
llat12	0.563	0.018
llat22	(0.009)	(0.47)
lmax11	0.686	0.355
lmax21	(0.001)	(0.09)
lmax12	0.616	0.530
lmax22	(0.004)	(0.01)
ln11	0.606	0.573
ln21	(0.003)	(0.004)
ln12	0.391	0.386
ln22	(0.05)	(0.05)
sdl11	0.283	0.229
sdl21	(0.15)	(0.20)
sdl12	0.412	0.293
sdl22	(0.06)	(0.13)

C

VARIABLE	CONTROL	R.DEP.
tanp11	0.808	0.469
tanp31	(0.001)	(0.03)
tanp12	0.868	0.379
tanp22	(0.001)	(0.06)
tnp111	0.736	0.577
tnp121	(0.001)	(0.01)
tnp211	0.827	0.358
tnp221	(0.001)	(0.09)
tnp112	0.867	0.308
tnp122	(0.001)	(0.11)
tnp212	0.796	0.395
tnp222	(0.001)	(0.05)
tlat11	0.386	0.410
tlat21	(0.06)	(0.06)
tlat12	0.389	0.461
tlat22	(0.06)	(0.03)
tmax11	0.844	0.754
tmax21	(0.001)	(0.001)
tmax12	0.858	0.412
tmax22	(0.001)	(0.04)
tn11	0.570	0.732
tn21	(0.005)	(0.001)
tn12	0.650	0.690
tn22	(0.001)	(0.001)
sdt11	-0.286	0.295
sdt21	(0.13)	(0.13)
sdt12	0.141	0.229
sdt22	(0.29)	(0.17)

Tables 1b) and c): showing the inter-session Pearson correlation, and exact probabilitiy in parentheses, of the variables to light and tone onset, respectively, for both groups in both sessions.

VAR	CONTROL	R.DEP.	E.DEP.	VAR	CONTROL	R.DEP.	E.DEP.
amp111	0.715	0.286	0.827	amp211	0.663	0.217	0.378
amp112	(0.001)	(0.11)	(0.01)	amp212	(0.001)	(0.18)	(0.20)
lat111	0.217	0.499	-0.881	lat211	0.192	0.440	0.736
lat112	(0.19)	(0.01)	(0.005)	lat212	(0.22)	(0.02)	(0.03)
ar111	0.776	0.190	0.607	ar211	0.684	0.333	0.607
ar112	(0.001)	(0.21)	(0.07)	ar212	(0.001)	(0.08)	(0.07)
ta11	0.746	0.055	0.464				
ta12	(0.001)	(0.41)	(0.15)				
lanp11	0.901	0.627	-0.214	lnp111	0.888	0.530	-0.090
lanp12	(0.001)	(0.002)	(0.32)	lnp112	(0.001)	(0.01)	(0.42)
lnp211	0.846	0.666	-0.180	llat11	0.681	0.899	-0.388
lnp212	(0.001)	(0.001)	(0.35)	llat12	(0.001)	(0.001)	(0.19)
lmax11	0.857	0.612	0.580	ln11	0.635	0.373	0.857
lmax12	(0.001)	(0.003)	(0.09)	ln12	(0.002)	(0.05)	(0.007)
tanp11	0.900	0.876	0.464	tnp111	0.867	0.930	0.757
tanp12	(0.001)	(0.001)	(0.15)	tnp112	(0.001)	(0.001)	(0.02)
tnp211	0.920	0.838	0.400	tlat11	0.740	0.778	-0.567
tnp212	(0.001)	(0.001)	(0.19)	tlat12	(0.001)	(0.001)	(0.09)
tmax11	0.838	0.705	0.750	tn11	0.802	0.830	0.500
tmax12	(0.001)	(0.001)	(0.03)	tn12	(0.001)	(0.001)	(0.13)
eeg111	0.235	0.227	0.829	eeg211	0.043	0.376	0.893
eeg112	(0.17)	(0.17)	(0.01)	eeg212	(0.43)	(0.05)	(0.004)
eeg311	0.253	0.660	0.786	sd111	0.095	0.136	0.236
eeg312	(0.15)	(0.001)	(0.02)	sd112	(0.36)	(0.29)	(0.30)
sdt11	0.380	0.439	0.306				
sdt12	(0.06)	(0.03)	(0.25)				

Table 2a): showing inter-condition Pearson correlations (Spearman for endogenous depressives) for all CNV and ERP variables, together with exact probabilities in parentheses, within session one.

VARIABLE	CONTROL	R.DEP.	VARIABLE	CONTROL	R.DEP.
amp121	0.300	0.044	amp221	0.178	0.432
amp122	(0.11)	(0.43)	amp222	(0.23)	(0.03)
lat121	0.653	0.195	lat221	0.822	0.224
lat122	(0.001)	(0.20)	lat222	(0.04)	(0.17)
ar121	-0.049	0.310	ar221	0.149	0.467
ar122	(0.42)	(0.09)	ar222	(0.27)	(0.02)
ta21	-0.148	-0.093			
ta22	(0.27)	(0.27)			
lanp21	0.738	0.726)	lnp121	0.737	0.719
lanp22	(0.001)	(0.001)	lnp122	(0.001)	(0.001)
lnp221	0.708	0.751	llat21	0.724	0.335
lnp222	(0.001)	(0.001)	llat22	(0.001)	(0.09)
lmax21	0.812	0.421	ln21	0.672	0.301
lmax22	(0.001)	(0.05)	ln22	(0.001)	(0.09)
tanp21	0.928	0.656	tnp121	0.898	0.477
tanp22	(0.001)	(0.003)	tnp122	(0.001)	(0.03)
tnp221	0.895	0.679	tlat21	0.868	0.738
tnp222	(0.001)	(0.002)	tlat22	(0.001)	(0.001)
tmax21	0.639	0.670	tn21	0.815	0.682
tmax22	(0.003)	(0.002)	tn22	(0.001)	(0.001)
eeg121	0.648	0.414	eeg221	-0.015	0.639
eeg122	(0.001)	(0.04)	eeg222	(0.48)	(0.002)
eeg321	0.560	0.732	sd121	0.215	0.309
eeg322	(0.006)	(0.001)	sd122	(0.22)	(0.12)
sdt21	0.998	0.522			
sdt22	(0.001)	(0.02)			

Table 2b): showing inter-condition Pearson correlations for all CNV and ERP variables, together with exact probabilities in parentheses, within session two.

greater instability, in that an inverse relationship is evident.. In marked contrast are the ERP responses. Very highly significant correlations are obtained for ERP measures to both light and tone onset between sessions. Comparing measures for the two conditions separately suggests, in the following correlations, a remarkable stability: non-incentive condition to lights N1-P300 ($r=0.779$, $p>0.001$), P300-N2 ($r=0.766$, $p>0.001$), baseline-P300 ($r=0.686$, $p>0.001$) and P300 latency ($r=0.421$, $p>0.05$) and in the incentive condition N1-P300 ($r=0.533$, $p>0.01$), P300-N2 ($r=0.539$, $p>0.01$), baseline-P300 ($r=0.616$, $p>0.004$) and P300 latency ($r=0.563$, $p>0.01$). ERPs emitted to tones in the non-incentive condition correlated as follows: N1-P ($r=0.736$, $p>0.001$), P-N2 ($r=0.827$, $p>0.001$), baseline-P ($r=0.844$, $p>0.001$) and P latency ($r=0.386$, $p>0.06$), and in the incentive condition were N1-P ($r=0.867$, $p>0.001$), P-N2 ($r=0.796$, $p>0.001$), baseline-P ($r=0.858$, $p>0.001$) and P latency ($r=0.389$, $p>0.06$). With the exception of P latency to tones, which just failed to reach significance, these results are interesting in the level and significance of correlation; especially so in comparison to those of the CNV.

Although the different conditions in this experiment may invoke different psychological sets on the parts of subjects, a comparison of the variables between conditions is considered relevent in that it illustrates the individual responsiveness to changes in conditions. Inter-condition correlations for controls present contrasting pictures within the two sessions. In session one, early and late peak negativity correlate significantly ($r=0.715$, $p>0.001$ and $r=0.663$, $p>0.001$ respectively) as well as the corresponding areas ($r=0.776$, $p>0.001$ and $r=0.684$, $p>0.001$ respectively) and total area ($r=0.746$, $p>0.001$). In marked contrast, in the second session, only the latency to early peak amplitude demonstrated a significant correlation ($r=0.653$, $p>0.001$). Again in contrast to the CNV measures, ERP activity demonstrated a strong intra-session association. In session one,

emitted activity to lights between conditions demonstrated the following correlations: N1-P300 (r=0.888, p>0.001), P300-N2 (r=0.846, p>0.001), baseline-P300 (r=0.857, p>0.01), and latency (r=0.681 p>001) and to tones; N1-P (r=0.867, p>0.001), P-N2 (r=0.920, p>0.001), baseline-P (r=0.838, p>0.001) and P latency (r=0.740, p>0.001). In session two, emitted activity to lights correlated between conditions as follows: N1-P300 (r=737, p>0.001), P300-N2 (r=708, p>0.001) baseline-P300 (r=0.812, p>0.001) and p300 latency (r=0.724, p>0.001); and to tone onset as follows: N1-P (r=0.898, p>0.001), P-N2 (r=0.895, p>0.001), baseline-P (r=639, p>0.003) and P latency (r=0.868, p>0.001).

Whilst correlational analysis determines the degree of association between elements, or individuals, comprising two sets of scores, it provides no information on the relationship between the measurements themselves. That is to say, that information is derived relating to the inter-group comparison based on intra-group similarities. To complement this, the Wilcoxon matched-pairs signed rank test was applied to investigate for the existance of differences in central tendancy. This test is reported because it, unlike the Student 't' test, normally applied to parametric data, makes no assumptions about normality which, in the case of clinical studies, makes it particularly applicable. Although, in fact, the parametric 't' test was also applied, only minor differences between the two approaches were obtained. Between sessions normals demonstrated a tendancy in both conditions for the CNV measures to reduce (see Table 3a), which reached significance for the early peak amplitude (Z=-2.656, p>0.008) in the non-incentive condition, and for the corresponding area to reduce and latency to lengthen in the incentive condition (Z=-2.175, p>0.03 and Z=-2.017, p>0.04 respectively). The same tendancy for ERp measures to change between sessions is also evident in both conditions; significant for latency to P300 peak to reduce to lights in the non-incentive condition (Z=-2.00, p>0.04) and for the baseline-P to tones to increase (Z=-2.722, p>0.002) in the

VARIABLE	CONTROL	R.DEP.	VARIABLE	CONTROL	R.DEP.
ampl11	-2.656	-0.635	arl12	-0.664	-0.075
ampl21	(0.008)	(0.53)	arl22	(0.51)	(0.94)
amp211	-0.566	-0.686	ar212	-1.408	-2.277
amp221	(0.57)	(0.49)	ar222	(0.16)	(0.02)
ampl12	-0.483	-0.402	tal1	-0.402	-1.087
ampl22	(0.63)	(0.69)	ta21	(0.69)	(0.28)
amp212	-1.127	-1.704	tal2	-0.885	-2.053
amp222	(0.26)	(0.09)	ta22	(0.38)	(0.04)
lat111	-0.958	-1.670	eegl11	-0.261	-0.201
lat121	(0.34)	(0.09)	eegl21	(0.79)	(0.84)
lat211	-1.590	-1.167	eeg211	-1.087	-1.429
lat221	(0.11)	(0.24)	eeg221	(0.28)	(0.15)
latl12	-2.017	-0.684	eeg311	-0.443	-1.167
latl22	(0.04)	(0.49)	eeg321	(0.66)	(0.24)
lat212	-0.020	-0.166	eegl12	-1.489	-0.261
lat222	(0.98)	(0.87)	eegl22	(0.14)	(0.79)
arl11	-2.175	-0.187	eeg212	-0.282	-1.006
arl21	(0.03)	(0.85)	eeg222	(0.78)	(0.31)
ar211	-0.483	-1.167	eeg312	-0.604	-0.871
ar221	(0.63)	(0.24)	eeg322	(0.55)	(0.38)

Table 3a): showing the Wilcoxon matched-pairs signed rank 'Z' value for both conditions between sessions for both controls and reactive depressives for the CNV variables (the associated exact probability is contained in parentheses.

incentive. Apart from the above exceptions, CNV measures within the conditions retained the same central tendancy between sessions. A non-significant tendancy to reduction in CNV variables was observed in session one between conditions, and to increase in session two. By contrast, ERP amplitude measures to both lights and tone demonstrate a significant tendancy to reduce and latency to increase from incentive to non-incentive conditions, which was more marked

VARIABLE	CONTROL	R.DEP.	VARIABLE	CONTROL	R.DEP.
tanp11	-0.355	-1.319	lanp11	-1.160	-1.533
tanp21	(0.72)	(0.19)	lanp21	(0.25)	(0.12)
tanp12	-1.551	-0.697	lanp12	-0.497	-1.241
tanp22	(0.12)	(0.49)	lanp22	(0.62)	(0.21)
tnp111	-0.497	-1.887	lnp111	-1.344	-1.836
tnp121	(0.62)	(0.06)	lnp121	(0.18)	(0.07)
tnp211	0.000	-0.672	lnp211	-0.213	-1.008
tnp221	(1.00)	(0.50)	lnp221	(0.83)	(0.31)
tnp112	-1.586	-1.349	lnp112	-0.663	-1.577
tnp122	(0.11)	(0.18)	lnp122	(0.51)	(0.11)
tnp212	-0.450	-0.379	lnp212	-0.114	0.762
tnp222	(0.65)	(0.70)	lnp222	(0.91)	(0.45)
tlat11	-1.244	-0.423	llat11	-2.00	-0.392
tlat21	(0.21)	(0.67)	llat21	(0.04)	(0.69)
tlat12	-0.510	-0.710	llat12	-0.105	0.767
tlat22	(0.61)	(0.48)	llat22	(0.92)	(0.44)
tmax11	-0.659	-0.114	lmax11	-1.373	-0.853
tmax21	(0.51)	(0.91)	lmax21	(0.17)	(0.39)
tn11	-0.240	-0.765	ln11	-0.308	-2.254
tn21	(0.81)	(0.44)	ln21	(0.76)	(0.02)
tn12	-0.426	-2.482	ln12	-0.414	-2.025
tn22	(0.67)	(0.01)	ln22	(0.68)	(0.04)
sdt11	-0.776	-0.142	sdl11	-0.710	-0.388
sdt21	(0.44)	(0.89)	sdl21	(0.48)	(0.70)
sdt12	-0.259	-1.293	sdl12	-1.647	-0.982
sdt22	(0.80)	80.20)	sdl22	(0.10)	(0.33)

Table 3b): showing the Wilcoxon 'Z' value for both conditions between sessions for both controls and reactive depressives for the variables following tone onset; (parentheses contain exact probabilities).

VAR	CONTROL	R.DEP.	E.DEP.	VAR	CONTROL	R.DEP.	E.DEP.
ampl11	-1.568	-1.587	-1.352	ar121	-0.936	-0.933	
ampl12	(0.12)	(0.11)	(0.18)	ar122	(0.35)	(0.35)	
amp211	-0.544	-0.946	-0.314	ar221	-0.765	-0.093	
amp212	(0.59)	(0.34)	(0.75)	ar222	(0.44)	(0.93)	
ampl21	-0.610	-0.905		ta11	-0.282	-0.635	-0.845
ampl22	(0.54)	(0.36)		ta12	(0.78)	(0.53)	(0.40)
amp221	-0.071	-0.302		ta21	-0.724	-0.149	
amp222	(0.94)	(0.76)		ta22	(0.47)	(0.88)	
lat111	-0.828	-0.402	-1.690	eegl11	-0.262	-0.044	-2.197
lat112	(0.41)	(0.69)	(0.09)	eegl12	(0.79)	(0.96)	(0.03)
lat211	-0.118	-1.067	-0.169	eeg211	-1.348	-0.056	-0.105
lat212	(0.91)	(0.29)	(0.87)	eeg212	(0.18)	(0.95)	(0.92)
lat121	-0.284	-1.941		eeg311	-0.523	-0.224	-0.507
lat122	(0.78)	(0.05)		eeg312	(0.60)	(0.82)	(0.61)
lat221	-1.219	-0.221		eegl21	-2.414	-0.262	
lat222	(0.22)	(0.82)		eegl22	(0.02)	(0.79)	
arl11	-0.694	-0.635	-1.352	eeg221	-0.893	-0.181	
arl12	(0.49)	(0.53)	(0.18)	eeg222	(0.37)	(0.86)	
ar211	-0.121	-1.083	-0.169	eeg321	-0.166	-0.080	
ar212	(0.90)	(0.28)	(0.87)	eeg322	(0.87)	(0.94)	

Table 3c): showing Wilcoxon 'Z' statistic for reactive depressives and controls in both sessions, and for endogenous in the first, between conditions; exact probabilities are shown in parentheses.

in the first session. Considering changes within session one, significant differences in central tendancy were obtained to tone onset for N1-P (Z=-2.221, p>0.03), P-N2 (Z=-3.053, p>0.002) and baseline-P (Z=-2.373, p>0.02) and to lights for N1-P300 (Z=-2.395, p>0.02), P300-N2 (Z=-2.395, p>0.02) and baseline-P300 (Z=-1.96, p>0.05). In the second session, a significant reduction in central tendancy was

VAR	CONTROL	R.DEP.	E.DEP.	VAR	CONTROL	R.DEP.
tanp11	-2.657	-2.940	-2.028	tanp21	-2.391	-2.275
tanp12	(0.008)	(0.003)	(0.04)	tanp22	(0.02)	(0.02)
tnpl11	-2.221	-3.103	-2.366	tnpl21	-1.965	-2.38[illegible]
tnpl12	(0.03)	(0.002)	(0.02)	tnpl22	(0.05)	(0.02)
tnp211	-3.053	-2.678	-1.690	tnp221	-2.223	-1.68[illegible]
tnp212	(0.002)	(0.007)	(0.09)	tnp222	(0.03)	(0.09
tlat11	-0.296	-0.676	-0.944	tlat21	-1.836	-0.82[illegible]
tlat12	(0.77)	(0.50)	(0.34)	tlat22	(0.07)	(0.41)
tmax11	-2.373	-2.367	-1.352	tmax21	-0.502	-1.37
tmax12	(0.02)	(0.02)	(0.18)	tmax22	(0.62)	(0.17)
tn11	-2.069	-1.067	-2.201	tn21	-1.917	-0.436
tn12	(0.04)	(0.29)	(0.03)	tn22	(0.05)	(0.66)
sdt11	-0.625	-0.544	-0.169	sdt21	-0.980	-0.639
sdt12	(0.53)	(0.59)	(0.87)	sdt22	(0.33)	(0.52)
lanp11	-3.030	-1.087	-1.014	lanp12	-1.586	-0.947
lanp12	(0.002)	(0.28)	(0.31)	lanp22	(0.11)	(0.34)
lnpl11	-2.395	-0.675	-1.183	lnpl21	-1.491	-0.155
lnpl12	(0.02)	(0.50)	(0.24)	lnpl22	(0.14)	(0.88)
lnp211	-2.395	-1.254	-0.845	lnp221	-1.887	-1.823
lnp212	(0.02)	(0.21)	(0.40)	lnp222	(0.06)	(0.07)
llat11	-1.608	-0.560	-0.105	llat21	-0.592	-0.866
llat12	(0.11)	(0.57)	(0.92)	llat22	(0.55)	(0.39)
lmax11	-1.960	-1.107	-0.592	lmax21	-2.223	-0.568
lmax12	(0.05)	(0.27)	(0.55)	lmax22	(0.03)	(0.57)
ln11	-0.402	0.302	-0.105	ln21	-0.450	-1.710
ln12	(0.69)	(0.76)	(0.92)	ln22	(0.65)	(0.09)
sdl11	-0.491	-1.852	-0.734	sdl21	-2.045	-1.086
sdl12	(0.62)	(0.06)	(0.46)	sdl22	(0.04)	(0.28)

Table 3d): showing the between condition Wilcoxon 'Z' values for the ERP variables for reactive depressives and normals for both sessions, and endogenous depressives in session two: (figures in parentheses denote exact probabilities).

observed to lights for baseline-P300 ($Z=-2.223$, $p>0.03$) and to tones only for N1-P ($Z=-1.965$, $p>0.05$) and P-N2 ($Z=-2.223$, $p>0.03$). In neither session were reductions in central tendancy of latencies observed. Although the relevence of these CNV and ERP results will be more fully evaluated in the discussion, it may here be posited that the ERP measures applied in this study appear to be more sensitive indicator than the CNV of a control subject's responsiveness to experimental manipulation and changes in this over time.

Before turning to the inter-session correlational analysis for reactive depressives, certain considerations may be introduced. Firstly, reliability indices for the measures with normals may be more meaningfully interpreted by comparison with a dissimilar group. Secondly, whilst changes in the measures may reasonably be expected for reactive depressives, the question remains open as to what degree such would be manifested by every member of the group. Significant positive correlations for reactive depressives were obtained between sessions in the incentive condition for late peak amplitude ($r=0.469$, $p>0.02$) and the corresponding area ($r=0.510$, $p>0.01$), and for latency to late peak negativity ($r=0.566$, $p>0.005$) in the non-incentive condition. Although, as with normals, significant positive correlations were more often encountered with ERP variables than CNV, those for reactive depressives were fewer in number and of reduced level in comparison. Significant correlations were obtained in non-incentive and incentive conditions to lights for N1-P300 ($r=0.484$, $p>0.03$ and $r=0.523$, $p>0.01$ respectively) and P300-N2 amplitudes ($r=0.468$, $p>0.03$ and $r=0.486$, $p>0.02$ respectively). Baseline-P300 amplitude just failed to reach significance ($r=0.355$, $p>0.09$) in the non-incentive, but was significant ($r=0.530$, $p>0.01$) under the monetary incentive, condition. P300 latency correlations to lights were not significant. In both non-incentive and incentive conditions to tones, base-

line-P correlations (r=0.754, p>0.001 and r=0.412, p>0.04 respectively) and latency (r=0.410, p>0.06 and r=0.412, p>0.03 respectively) proved significant. However, N1-P in the non-incentive condition produced a strong association (r=0.577, p>0.01) as did P-N2 in the incentive (r=0.395, p>0.05).

Within sessions, the overall picture for CNV variables appears different to that of normals and that of ERP variables strikingly similar. In session one, a significant correlation was obtained for both latency measures (early: r=0.499, p>0.01 and late: r=0.440, p>0.02). In session two, only late maximum amplitude of the CNV correlated between conditions (r=0.467, p>0.02). However, ERP measures again demonstrated a remarkable association. In session one, the following correlations to light onset between non-incentive and incentive conditions were observed: N1-P300 (r=0.530, p>0.01), P300-N2 (r=0.666, p>0.001), baseline-P300 (r=0.612, p>0.003) and P300 latency (r=0.899, p>0.001); and to tone onset, N1-P (r=0.930, p>0.001), P-N2 (r=0.838, p>0.001), baseline-P (r=0.705, P.001), and latency to peak positivity (r=0.778, p>0.001). In the second session, the correlations obtained were as follows; to lights: N1-P300 (r=0.719, p>0.001), P300-N2 (r=0.751, p>0.001), baseline-P300 (r=0.421, p>0.05), but P300 latency failed to reach significance (r=0.335, p>0.09); and to tone onset: N1-P (r=0.477, p>0.03), P-N2 (r=0.679, p>0.002), baseline-P (r=0.670, p>0.001) and P latency (r=0.738, p>0.001).

The Wilcoxon scores for reactive depressives demonstrate a dissimilar picture in central tendancy to controls of increasing amplitudes and shortening latencies between sessions. A significant difference was observed in the incentive condition for late (Z=-2.277, p>0.02) and total (Z=-2.053, p>0.04) CNV areas. Within session one, a non-significant trend for a reduction in amplitudes is observed. In session one, a significant reduction is observable in the ERP to tone onset in the N1-P (Z=-3.103, p>0.002), P-N2 (Z=-2.678, p>0.007), and baseline-P (Z=-2.367, p>0.02) but

not in P latency, nor any CNV, nor ERP to lights, measures. In the second session, a significant reduction in central tendancy between conditions was obtained only for latency to early peak CNV amplitude ($Z=-1.941$, $p>0.05$) and to N1-P amplitude to tone onset ($Z=-2.385$, $p>0.02$).

Reference to Tables 2 and 3 suggests that a separate process, exerting an independent effect, is involved, in that N1-P300 amplitude does not exactly match variations in the baseline-to-peak positivity measures. To assess such an N1 effect, the latter measure was subtracted from the former and the intra-, and inter-,session correlations, as well as differences in central tendancy, calculated; these statistics are included in the appropriate tables. Control subjects, within sessions, demonstrated a very high association between scores to lights in session one ($r=0.635$, $p>0.002$) and session two ($r=0.672$, $p>0.001$) and to tone onset ($r=0.802$, $p>0.001$ and $r=.615$, $p>0.001$ respectively). Between sessions, negativity to lights demonstrated also a significant relationship (non-incentive:$r=0.606$, $p>0.003$; incentive:$r=0.39$), $p>0.05$). Highly significant associations between sessions were also obtained in non-incentive ($r=0.57$, $p>0.001$) and incentive ($r=0.65$, $p>0.001$) conditions for normals to tone onset. The same trend of reducing amplitude across both conditions and sessions, evident for the other ERP variables in the Wilcoxon statistic, holds strongly for N1 negativity. This produced a significant difference to tones in both sessions (session 1: $Z=-2.069$, $p>0.04$; session 2: $Z=-1.917$, $p>0.05$), but not across sessions, nor to lights. Reactive depressives demonstrated a similar pattern in their within-session correlations to N1 to tone onset (session one: $r=0.830$, $p>0.001$ and session two: $r=0.682$, $p>0.001$). To the onset of lights, a significant correlation, and that of a markedly lower level, was obtained only in the first session. Between sessions, significant correlations were obtained in both non-incentive (to lights: $r=0.573$, $p>0.004$;tone: $r=0.732$, $p>0.001$) and incentive (to lights: $r=0.386$, $p>0.05$; to tone: $r=0.690$, $p>0.001$) condit-

ions. Whereas controls demonstrated a significant reduction in N1 negativity between conditions to tones in both sessions, reactive depressives did not. Interestingly, no reduction was observed between conditions in either session, but rather a reduction between sessions to light onset ($Z=-2.254$, $p>0.02$) in the non-incentive condition and, in the incentive condition, to both light ($Z=-2.025$, $p>0.04$) and tone ($Z=-2.482$, $p>0.01$) onset. These significant reductions in N1 amplitude over time suggest it to be an important indicator of subjective performance.

Finally, in this presentation of the reliability measures of the ERP concerning controls and reactive depressives, attention must be given to the standard deviation, around the measures in the raw data for each subject. Of concern is whether variability itself presents an enduring and distinctive individual characteristic. Dealing first with between session correlations and comparisons of central tendancy, controls demonstrated a high correlation in early ($r=0.770$, $p>0.001$, middle ($r=0.618$, $p>0.002$) and late variability ($r=0.502$, $p>0.01$) in the CNV. No significant correlations were obtained in positivity to either light or tone onset. These measures produced no significant correlation in session one, but in session two early and late CNV variability, between conditions, correlated ($r=0.648$, $p>0.001$ and $r=0.560$, $p>0.006$ respectively), as did variability in positivity to tone onset ($r=0.998$, $p>0.001$). No significant differences in central tendancy were observed between sessions, or within session one. Only in session two were significant reductions in early CNV variability ($Z=-2.414$, $p>0.02$) and in positivity to lights ($Z=-2.045$, $p>0.04$) recorded. Correlations in variability measures, in reactive depressives, presented the following significant statistics. Between sessions, in the non-incentive condition, early CNV variability was significantly correlated ($r=0.727$, $p>0.001$). Correlating non-incentive and incentive conditions in session one produced significant results for late CNV variability ($r=0.660$, $p>0.001$) and positivity variability to

tones ($r=0.439$, $p>0.03$), and in session two for early ($r=0.414$, $p>0.04$), middle ($r=0.639$, $p>0.02$) and late ($r=0.732$, $p>0.001$) CNV variability and for variability in positivity to tone onset ($r=0.439$, $p>0.03$). For neither group did variability to light onset correlate either between conditions or sessions. No significant trends in variability measures were noted either within, or between, sessions for reactive depressives, although, as with the other variables, the tendancy was for a reduction in values recorded.

Although only recorded on admission to hospital, and concerning only a small number of patients, observations can be made on the between condition correlations and changes in central tendancy for endogenous depressives. For this group, the nonparametric 'r' is reported. Significant correlations were obtained for early CNV amplitude ($r=0.827$, $p>0.01$), an inverse correlation with the associated latency ($r=-0.881$, $p>0.005$) and a positive correlation with late peak latency ($r=0.736$, $p>0.03$). The associated variability in early, middle and late CNV amplitude correlated significantly ($r=0.829$, $p>0.01$; $r=0.893$, $p>0.004$; $r=0.786$, $p>0.02$ respectively). Of the ERP measures, to light onset, only N1 negativity produced a significant correlation between these conditions ($r=0.857$, $p>0.007$). To tone onset, significant correlations were obtained to N1-P ($r=0.757$, $p>0.02$) and baseline-P ($r=0.750$, $p>0.03$), but to none other. Significant reductions in central tendancy were observed only to the ERP measures to tone onset in the N1-P ($Z=-2.366$, $p>0.02$) and N1 negativity ($Z=-2.201$, $p>0.03$). A clear implication here is that the former may well be largely accounted for by the latter, in that baseline-P demonstrated no change in central tendancy.

Without pre-empting a full discussion later, certain considerations may be introduced at this stage. Apparent in the results is the absence of any pattern, which suggests that not only do the CNV and ERP measures reflect different processes, invoked by variation within the experimental

setting, but that the CNV itself represents no singular process. However, certain conclusions can be offered to the posited hypotheses. Taking firstly the correlational results, indicating the stability of scores for the individual subject within their group, retest reliability is suggested only for late CNV area in the incentive condition and early area in the non-incentive. Between conditions, however, a clear association is indicated, excepting latency, in all measures in the first session, and only in latency to early peak amplitude in the second. Whilst the second session appears anomalous, the results obtained indicate a short-term test-retest reliability, but not a long term. ERP measures to lights and tone onset demonstrate more consistent correlations in both between condition comparisons in sessions one and two, as well as between the sessions themselves. Introducing the results on measures of central tendancy, which reflects the stability of the scores, rather than that score's position within a group, a significant reduction is indicated between sessions in early CNV amplitude and the associated area in the non-incentive condition. ERP measures, to both light and tone onset in session one, and to tone onset in session two, demonstrate consistent reductions in scores. Accordingly, lacking clear-cut support, hypothesis one is refuted in that both early and late CNV amplitudes and areas vary in the long-term, and covary in the short, with the suggestion of significant reductions in early, rather than late, CNV amplitude and area between sessions. Concerning solely the comparison of central tendancy between conditions for normals, no significant reduction in late positivity latency to either tone or light onset is observed: both just failing to reach significance in session two. A significant reduction in late positivity is suggested by the baseline-to-peak positivity measure to both lights and tones in session one and to tones in session two, by both negativity-to-peak positivity measures in both sesions to tones, and by the negativity-to-peak positivity measure to lights in session one. Accordingly, hypothesis

six is refuted.

5.3. Group differences.

To evaluate group differences, the Mann-Whitney statistic was applied to the averaged EEG data. To enable statistical comparisons with endogenous depressives in session one, comparison samples from controls and reactive depressives, selected to equate age and sex as far as possible, were formed. Although, the Student 't' test was also performed, the Mann-Whitney 'U' statistic is here reported, in that it makes no assumptions about normality of the data, which is basic to the t-test. Although 't' is not reported, it may be noted that neither were additional variables, demarkating the two groups, identified, nor was the level of significance, obtained by the Mann-Whitney, radically altered for the sensitive variables identified through the application of the 't' statistic in the evaluation of group differences. The relevent tables may be found in Appendix G.

Dealing, firstly, with the comparison of controls with reactive depressives in the first session, no significant differences were obtained for CNV or ERP variables, or their standard deviations, in the non-incentive condition. Within the incentive condition of session one, only N1 negativity to tones discriminated the two groups (U=115.0, p>0.03). Although both groups, in contrast to the non-incentive condition, demonstrate a reduction in this variable, this was more marked for normals (normals: 12.04 to 10.05 μv; reactive depressives: 15.17 to 14.83 μv). This absence of group differences is also apparent in session two. In both non-incentive and incentive conditions, no variable significantly separated the two groups. The variables negativity to lights (U=136.0, p>0.07) in the non-incentive, and, in the incentive condition, baseline-maximum positivity to lights (U=105.5, p>0.08) and early CNV variability (U=130.0,

$p>0.09$) just failed to reach significance. The overriding impression, obtained from the inspection of these tables, is one of an absence of differences between the groups in the CNV and ERP variables in both conditions of both sessions.

The comparison of endogenous depressives against controls also failed to identify clear-cut group differences. Of the CNV variables, only early CNV standard deviation in the non-incentive condition ($U=8.0$, $p>0.03$) produced significant group differences; with depressives demonstrating lower (8.47 μv as against 11.48 μv) mean standard deviation of amplitude. In the non-incentive condition, latency to late peak amplitude ($U=11.5$, $p>0.09$; mean controls: 11.1 sec.; mean depressives: 10.82 sec) and, in the incentive condition, to early peak amplitude ($U=11.5$, $p>0.09$; mean controls: 7.12 sec; mean depressives: 9.09 sec) just failed to obtain the cut-off significance level. In the incentive condition, comparison of the two groups for the ERP variables produced a significant difference only for N1-P amplitude to tone onset ($U=9.0$, $p>0.05$; mean controls: 10.05; mean depressives: 15.46 μv). In the non-incentive, following tone onset, N1 negativity ($U=3.0$, $p>0.006$; mean controls: 12.64 μv; mean depressives: 19.96 μv) and N1-P amplitude ($U=9.0$, $p>0.04$; mean controls: 23.89 μv; mean depressives: 30.13 μv) demonstrated significant group differences. However, comparison of mean differences, and that baseline-P amplitude did not discriminate between the groups strongly suggests that the latter significant difference may be accounted for by the first. Significant group differences, in the non-incentive condition, were also obtained for latency to maximum positivity following tone onset ($U=10.0$, $p>0.05$; mean controls: 214.44 msec; mean depressives: 222.86 msec). No significant group differences obtained to light onset, nor in any standard deviation measure of the ERP variables.

Finally, endogenous depressives were compared against reactive for both CNV and ERP variables. As with controls, standard deviation in the early part of the CNV, in the

non-incentive condition, produced significant differences between endogenous and reactive depressives (U=8.0, p>0.03; mean endogenous: 8.47 μv; mean reactive: 11.88 μv). Otherwise, comparison of the CNV variables produced no significant group differences in either incentive or non-incentive conditions. In contrast to the comparison with normals, N1 negativity and N1-P amplitude to tones did not discriminate between the groups, but latency to maximum positivity to tone onset, in the non-incentive condition, did (U=6.0, p>0.02; mean endogenous: 222.86 msec; mean reactive: 253.33 msec). Further, to light onset in the non-incentive condition, P300-N2 amplitude (U=6.0, p>0.02; mean endogenous: 16.77 μv; mean reactive: 19.19 μv) and the standard deviation to maximum positivity (U=8.5, p>0.04; mean endogenous: 10.57 μv; mean reactive: 14.25 μv) produced significant differences between these groups. One observation that can be made at this stage is the striking absence of group differences in the incentive condition, in contrast to the non-incentive, which clearly suggests that, within this experimental paradigm, both groups of depressives could fulfill the task requirements equally as well as controls.

Applying the above results to the hypotheses presented permits the following conclusions. Although there is some suggestion of lower CNV amplitudes and delayed latencies (see Appendix C) in the comparison of reactive depressives and controls, these differences are not significant. Again not reaching significance, endogenous depressives demonstrate lower CNV amplitudes and delayed peak latencies in comparison to controls. Accordingly, hypothesis two must be rejected in that CNV variables do not permit a separation of the three groups at the first recording. In the light of no significant differences between reactive depressives and controls, on the former's discharge from hospital, hypothesis three must be accepted.

Evaluation of the group comparisons for the ERP data permits the following conclusions. Although maximum positivity to tones and lights, as well as the associated to

peak-to-peak measures, suggest reduced positivity amplitude in the comparison of reactive depressives and controls, these differences do not attain acceptable significance levels. Similarly, differences in latency to maximum positivity, following light and tone onset, do not reach acceptable significance levels in either condition. Considering endogenous depressives, these demonstrated earlier peak positivity in comparison to both reactive depressives and controls in the non-incentive condition. Since, as mentioned above, differences in peak-to-peak amplitude may be accounted for by the contribution from N1 negativity, then the only significant group differences were for P300-N2 amplitude to light onset and the standard deviation to maximum positivity between endogenous and reactive depressives. In summary, these results are not consistent with the direction of differences posited in hypothesis four which is, therefore, rejected. On discharge from hospital reactive depressives demonstrated non-significant peak-to-peak positivity amplitude and maximum positivity differences to controls to tone onset, although the data indicates a reduction. Similarly, a non-significant delay in peak latency is suggested. Baseline-to-P300 positivity to light onset does discriminate between groups, in the non-incentive condition, although peak-to-peak positivity measures do not. Accordingly, hypothesis five can be accepted, but only insofar as ERP measures to tone onset are concerned.

5.4. Behavioural measures.

Whilst not of central concern to the present study of electrocortical events associated with changes in incentive conditions in reactive depressives, the association between CNV and ERP measures and certain behavioural data is of interest. The behavioural data to be introduced here concerns average reaction time in each condition, the Befindlichkeitskala (Bfs) and Beck scale, and the self

rating of performance. Firstly, the correlation of these measures with those extracted from the EEG, and then inter-group comparisons will be reported. Tables are presented in Appendices H and I for these analyses respectively. All subjects, in each group, were solely, or predominantly, right-handed.

Average reaction time demonstrated no correlation in either condition of session one for controls with any of the CNV measures used. In the second session, this group demonstrated a negative correlation ($r=-.387$, $p>0.05$) in this measure with late area of the CNV in the non-incentive condition, and a positive correlation ($r=.427$, $p>0.03$) with early area in the incentive condition. Reactive depressives produced only a correlation ($r=0.417$, $p>0.03$) between average reaction time and the CNV measure of early area, in the incentive condition of session one. Normals demonstrated negative correlations, in session one in the non-incentive condition, with N1-P amplitude ($r=-0.343$, $p>0.08$) and P-N2 ($r=-0.367$, $p>0.07$), and, in the incentive condition, with these two measures ($r=-0.585$, $p>0.005$ and $r=-0.557$, $p>0.008$ respectively) and with baseline-to-maximum positivity ($r=-0.537$, $p>0.01$). Generally this feature, of a higher correlation with ERP measures to tone onset in the incentive condition, albeit often inverse, is apparent for both groups in both sessions. A negative correlation, for controls, is also evident in the incentive condition of session two, in the ERP to tone onset, for P-N2 amplitude ($r=-0.441$, $p>0.04$). An association in ERP measures to light onset is observed only in session two, and then in both conditions (non-incentive: $r=0.499$, $p>0.02$; incentive: $r=0.343$, $p>0.09$). Whereas normals demonstrated significant associations between ERP amplitude measures to tones and average reaction time, reactive depressives demonstrated a different pattern. Although, in session one, no correlation in the non-incentive condition was observed, in the incentive condition a correlation with latency to peak positivity following tone onset was observed ($r=0.493$, $p>0.02$). This

appeared also in the non-incentive condition of session two with positive correlations following tone onset ($r=0.515$, $p>0.002$) and light ($r=0.407$, $p>0.05$) being obtained. In the incentive condition, reactive depressives demonstrated no further significant correlations with latency measures, but did with amplitude. A negative correlation obtained with N1-P amplitude to tone onset ($r=-0.420$, $p>0.04$), and positive with N1-P300 amplitude ($r=0.452$, $p>0.03$) and baseline-to-peak positivity ($r=0.489$, $p>0.02$) following light onset. The pattern of correlation changes for the two groups so that any strict relationship between average reaction time and any CNV or ERP measure is difficult to identify. That such a relationship could only be tenuous is further supported in that reactive depressives, prior to discharge from hospital, did not depict any pattern of correlations typical of normals in either recording session.

Both the CNV and Bfs are reported as sensitive to subject's present mood. However, an inspection of the correlation supports no clear relationship. A correlation, in controls in session one, was seen with latency to late peak amplitude ($r=0.380$, $p>0.05$) in the non-incentive, and inversely with early peak latency ($r=-0.641$, $p>0.002$) in the incentive, condition. This relationship was also apparent in session two; in which early peak latency correlated in the non-incentive condition ($r=0.438$, $p>0.03$) and in the incentive ($r=420$, $p>0.04$). No significant relationship with amplitude measures was observed. By contrast, reactive depressives demonstrated no correlations with the Bfs in the non-incentive condition of either session one or two. Only in the incentive condition to session two, and not in session one, are negative correlations observed; and these with late peak amplitude ($r=-0.415$, $p>0.05$) and total area ($r=-0.505$, $p>0.02$). The aforementioned suggestion that the CNV and ERP measures reflect different processes, is further supported here in the large-scale absence of correlations in the latter with the Bfs. Normals demonstrated, in the incentive condition of session one, a positive correlation

with latency of peak amplitude to tone onset ($r=0.414$, $p>0.04$), and, to light onset, a negative correlation with N1 negativity ($r=-0.490$, $p>0.02$). Reactive depressives, in the non-incentive condition of session two, demonstrated an inverse correlation with N1-P amplitude ($r=-0.561$, $p>0.02$), and, in session one, with N1 negativity ($r=-0.574$, $p>0.006$). Although a negative correlation between the Bfs and amplitude measures is intuitively acceptable, why it should prevail for reactive depressives in session two, and not for controls in either, is not clear, but cannot be explained as a statistical artifact in that the range of scores for both groups was not reduced between sessions. Similarly, both positive and inverse correlations between latency measures and the Bfs was also difficult to contain in a single descriptive statement.

No greater consistency is evident in the correlations between the Beck Depression Scale and the CNV and ERP measures obtained. It should be noted that, for controls, the Beck Scale was assessed only in session one. Whilst controls demonstrated predominantly inverse correlations between their Beck Scale rating and CNV measures, these reached significance only in the non-incentive condition with early peak amplitude ($r=-0.417$, $p>0.04$) and, in the incentive, with latency to early peak amplitude ($r=-0.450$, $p>0.03$). No significant correlations were observed with ERP variables. Reactive depressives demonstrated, interestingly, positive correlations in the non-incentive condition of session one which reached significance for the measures of early peak amplitude ($r=0.477$, $p>0.02$), and the corresponding area ($r=0.516$, $p>0.01$) and total area ($r=0.528$, $p>0.01$). That a high depression score should be related to high negativity mesures in the CNV will be more fully discussed later. In the incentive condition, negative correlations obtained with both latency to early and late peak negativity ($r=-0.515$, $p>0.01$ and $r=-0.400$, $p>0.04$ respectively). A negative correlation, in the non-incentive condition, with the ERP measures of N1 negativity ($r=-0.472$,

$p>0.02$) to light onset was observed. In the incentive condition, positive correlations obtained with P-N2 amplitude ($r=0.455$, $p>0.03$) and baseline-to-maximum positivity ($r=0.556$, $p>0.01$) to tone onset; but none were observed to light onset. In neither condition of session two did reactive depressives demonstrate significant correlations between CNV variables and their Beck score. With the ERP measures, a positive correlation with N1-P amplitude ($r=0.516$, $p>0.04$) in the non-incentive, and a negative in the incentive ($r=-0.539$, $p>0.02$), conditions to tones were obtained; neither baseline-to-peak nor N1 negativity alone produced significant correlations. The only correlation to lights, for reactive depressives in session two, was with baseline-P300 positivity in the non-incentive condition ($r=0.472$, $p>0.04$).

Concerning the association between these behavioural variables, certain relationships might be expected as well as the ability of such to discriminate between the groups. Thus psychomotor retardation, as expressed in longer reaction times, might be expected to correlate with either, or both of, the Beck or Bfs scales. Although these obtained correlations did not approach significance, normals tended to demonstrate an inverse correlation between these variables (see Appendix I). Reactive depressives demonstrated a significant correlation in the expected direction only in the non-incentive condition of session two with the Beck scale ($r=0.430$, $p>0.04$). This obtained although the scores of reactive depressives' correlations between Beck and Bfs were significant on admission ($r=0.646$, $p>0.002$) and discharge ($r=0.816$, $p>0.001$) from hospital. Highly significant correlations were also obtained, in session one between these measures, both for controls ($r=0.652$, $p>0.001$) and endogenous depressives ($r=0.800$, $p>0.05$).

As with the EEG measured, no strict relationship between correlations and central tendancy was observed for the variables average reaction time, Bfs and Beck scales. Normals demonstrated a highly significant correlation in the

Bfs between sessions (r=0.561, p>0.007) but no differences in central tendancy were observed. Similarly, reactive depressives demonstrated a high inter-session correlation coefficient (r=0.688, p>0.001) with no significant trend in central tendancy. Over this period, the latter group also demonstrated high intra-group stability (r=0.532, p>0.01) but with a significant reduction in the Beck score (Z=-3.15, p>0.002; mean session one: 29.00; mean session two: 14.69). This result underlines the point that Bfs and Beck scales have different referents.

Both normals and reactive depressives demonstrate high inter-session correlations in average R.T. for both conditions (normals in condition one: r=0.643, p>0.001; in conditiion two: r=0.725, p>0.001; reactive depressives in condition one: r=0.443, p>0.02; in condition two:r=0.701, p>0.001). That no differences in central tendancy were observed clearly indicates that both normals and depressives performed similarly under both conditions within sessions. To which needs to be added the significance of inter-condition differences in both sessions between the two groups, which suggests that both performed, and modified their performance to changes in experimental conditions, similarly as individuals within their groups. That controls uniformly responded in average R.T. to the introduction of incentives is indicated by the highly significant correlations and shifts in central tendancy in both sessions (session one:r=0.904, p>0.001;Z=-2.82, p>0.005; mean condition one:303.79 msec, mean condition two:278.21 msec:session two r=0.893, p>0.001;Z=-3.44, p>0.001; mean condition one:288.5 msec; mean condition two:264.1 msec). Reactive depressives responded similarly, except that no differences in central tendancy between conditions in session two were observed (session one:r=0.714, p>0.001;Z=-2.70, p>0.007; mean condition one:274.6 msec, mean condition two:238,25 msec: session two: r=0.839, p>0.001; z=-1.70, p>0.09;mean condition one:248.7 msec,mean condition two 248.15 msec). Comparable also were endogenous depressives in session one (r=0.893,

p>0.004;Z=-2.37, p>0.02; mean session one:276.57, mean session two:249.86 msec).

Mann-Whitney analysis was applied to determine whether differences between the groups, on the basis of these measures, could be determined. Neither average reaction time nor satisfaction with performance in either condition, or in either session, discriminated between controls and either patient group or between reactive and endogenous depressives. Significant differences were obtained in the Bfs between controls in session one and reactive depressives (U=38, p>0.0001;mean controls:13,32, mean reactive depressives:29.83) and endogenous depressives (U=8.5, p>0.07; mean controls:11.86, mean engogenous depressives:26.71) and in session two between controls and depressives (U=54.5, p>0.002; mean controls: 12.22, mean depressives: 29.19). Endogenous depressives were not distinguishable from reactive on the basis of the Bfs. Beck score differences between the groups were found to be significant in the following instances; between reactive depressives and controls(U=0.000, p>0.000; mean controls:2.42, mean depressives:29.00), between endogenous depressives and controls (U=0.000, p>0.007, mean controls: 2.00, mean depressives: 19.20) and between the depressive groups (U=2.0, p>0.05; mean reactive depressives:25.71, mean endogenous depressives:19.20).

Turning, finally, to reporting the analysis of the self-rating scale, only its relationship with average reaction time and the Bfs and Beck scales will be presented. Normals demonstrated non-significant negative correlations obtained in session one with average reaction time under either condition and with the Bfs. A significant negative correlation did obtain with the Beck scale (r=-0.410, p>0.04) for normals. These, in session two, produced a negative correlation with the Bfs (r=-0.566, p>0.007); but again no association was observed with average reaction time. Reactive depressives demonstrated no significant correlations between these measures and the self-rating scale in session one; but in session two negative correl-

ations were observed with all three (Beck:r=-0.577, p>0.01; Bfs:r=-0.416, p>0.05; average reaction time in condition one: r=-0.369, p>0.06; in condition two:r=-0.396, p>0.05). A negative correlation between performance self-rating and average reaction time is to be expected, in that the higher the self-rated score the faster is reaction time, i.e. the lower its absolute value. Whilst the level of these correlations is not high, that they did not also obtain for normals is suprising. Inter-session self-ratings showed a non-significant reduction (controls: Z=-1.609, p>0.11; reactive depressives: Z=-0.899, p>0.37). No group differences at all were obtained for this measure of performance.

5.5. Multivariate analysis of the data.

This analysis is introduced on two grounds. Firstly, multivariate statistical analysis is appropriate where multiple sources of influence are associated with multiple outcomes. The former, in this instance, consist of the presence, and severity of, depressive symptoms and their responsiveness to experimental manipulation in comparison to a control group. The latter consist of the CNV and ERP measures. The measurement of such variables constitute a model of a behavioural construct which is to be evaluated in its ability to describe the construct of interest. In this instance, interest is focused on whether the measurement model for reactive depressives is distinct to, and consequently distinguishable from, that of normal controls (Finn 1974). Secondly, no simple means exist to discriminate bewteeen the CNV and ERP variables as to their ability to describe the whole phenomenon or to isolate some distinctive critical sensitivity. All of the variables studied can vary in response to, for instance, variation in experimental conditions. Further, the analysis of such variables as are highly intercorrelated, as is probably the case with CNV and ERP data, results in the loss of information contained in

VARIABLE	UNIVAR.F	P<	STEP DOWN	P<
amp1xc	.660	0.422	.660	0.422
amp2xc	.264	0.610	.037	0.848
lat1xc	.007	0.935	.020	0.889
lat2xc	.038	0.845	.070	0.792
ar1xc	1.677	0.204	1.422	0.242
ar2xc	1.700	0.201	2.746	0.108
taxc	1.399	0.245	1.366	0.252
amp1xs	.369	0.547	.141	0.710
amp2xs	.525	0.473	.017	0.897
lat1xs	.734	0.397	.126	0.725
lat2xs	1.477	0.232	5.394	0.028
ar1xs	.563	0.458	.305	0.586
ar2xs	1.271	0.267	2.136	0.157
taxs	1.265	0.268	.006	0.941
amp1xcxs	1.862	0.181	3.076	0.093
amp2xcxs	.537	0.468	6.522	0.018
lat1xcxs	3.621	0.065	2.405	0.137
lat2xcxs	1.149	0.291	3.373	0.082
ar1xcxs	1.522	0.225	.001	0.975
ar2xcxs	.052	0.821	.291	0.597
taxcxs	.153	0.698	1.478	0.242

F-statistic for multivariance test of equality of mean vectors (F-approxiamation ratio)= 1.964. D.F.:21 and 16. $p < 0.086$.

Table 4a): showing univariate and step down F values of CNV variables, and the corresponding p, and their interaction with condition and session in the 'natural order'.

the variability of the individual measures, which is highly inter-related between them. The multivariate approach is applied to the data set as a whole and, as such, analyses multiple sources as a set of interrelated descriptors. In this experiment, subjects were classified on factor one with

B

VARIABLE	STEP DOWN	P≤
latlxcxs	3.621	0.065
amp2xcxs	1.023	0.319
lat2xs	1.185	0.284
lat2xcxs	4.800	0.035
amplxcxs	2.870	0.100
amplxc	.566	0.457
amp2xc	1.039	0.316
latlxc	.093	0.763
lat2xc	.598	0.446
arlxc	1.201	0.283
ar2xs	7.691	0.010
taxs	2.224	0.148
amplxs	.755	0.393
amp2xs	.345	0.563
latlxs	.001	0.972
arlxs	.249	0.623
ar2xs	5.882	0.025
taxs	.170	0.685
arlxcxs	.001	0.975
ar2xcxs	.291	0.597
taxcxs	1.477	0.242

C

VARIABLE	STEP DOWN	P≤
amplxc	.660	0.422
amp2xc	.037	0.848
latlxc	.020	0.889
lat2xc	.070	0.792
arlxc	1.422	0.242
ar2xc	2.746	0.108
taxc	1.366	0.252
amplxs	.141	0.710
amp2xs	.017	0.897
latlxs	.126	0.725
arlxs	.000	0.998
ar2xs	1.263	0.272
taxs	.022	0.884
arlxcxs	.887	0.356
ar2xcxs	6.945	0.015
taxcxs	.053	0.820
amplxcxs	.866	0.363
lat2xcxs	.864	0.364
lat2xs	5.817	0.027
amp2xcxs	1.492	0.239
latlxcxs	4.623	0.047

Tables 4b) and c): showing step down F values for between group differences when variables of interest are placed at the begining, or the end, of variables input to the analysis; univariate F values are unaffected (see Table 4a).

three levels crossed with a repeated measure analysis-of-variance design (level one: session, level two: condition, level three: parameters).

Prior to the application of multivariate analysis (Finn 1977), the data matrices for CNV and ERP data for each group

VARIABLE	1	2	3	4	5	6
amp1xc	.652	.043	-.439	.160	.016	.445
amp2xc	.569	.273	-.529	-.353	-.223	-.273
lat1xc	.293	-.330	-.099	-.011	.524	.228
lat2xc	-.117	-.253	-.276	-.047	-.036	.015
ar1xc	.699	.089	-.449	.319	.270	.256
ar2xc	.591	.291	-.517	-.365	-.201	-.188
taxc	.752	.219	-.557	.153	.063	.030
amp1xs	-.046	-.687	-.330	.536	.197	.097
amp2xs	-.227	-.700	-.327	-.390	-.303	-.003
lat1xs	-.239	-.174	.260	-.581	.408	.191
lat2xs	-.260	-.255	-.176	.431	-.558	.056
ar1xs	-.243	-.714	-.282	.350	.311	-.044
ar2xs	-.225	-.709	-.308	-.405	-.266	-.114
taxs	-.286	-.860	-.340	-.031	.060	-.095
amp1xcxs	-.491	.445	-.508	-.001	.264	-.090
amp2xcxs	-.750	.335	-.264	.183	.073	.243
lat1xcxs	-.262	-.023	-.318	-.473	-.116	.469
lat2xcxs	-.036	-.054	-.141	-.614	.429	.295
ar1xcxs	-.583	.514	-.448	-.096	.119	-.343
ar2xcxs	-.800	.370	-.313	.118	-.031	.094
taxcxs	-.776	.435	-.344	.133	.023	-.100

Table 4d): showing, reduced to three decimal places, the first six principle component loadings of the CNV variables input to multivariate analysis-of-variance.

were subjected to Joreskog factor analysis for the two conditions within each session separately. 'Jfactor', in comparison to the conventional factor analysis option of SPSS (Nie et al. 1975), offers the advantage of providing the Bartlett test of sphericity, the anti-image test, and the Kaiser-Meyer-Olkin measure of sampling adaquacy. This was pursued so that the suitability of the data matrix for factoring could be assessed. The Bartlett test has as the associated hypothesis that the correlation matrix comes from

VARIABLE	UNIVAR.F	P<	STEP DOWN	P<
N1	3.011	0.092	3.011	0.092
P-N2	.129	0.721	2.449	0.127
Plat	.046	0.831	.413	0.525
P	1.854	0.183	1.128	0.297
N1xc	1.463	0.235	1.185	0.285
P-N2xc	1.267	0.268	.682	0.416
Platxc	.474	0.496	.028	0.867
Pxc	.138	0.713	2.679	0.114
N1xs	4.05	0.044	1.723	0.201
P-N2xs	.146	0.705	.007	0.935
Platxs	.981	0.329	1.174	0.290
Pxs	1.738	0.196	13.599	0.001
N1xcxs	.147	0.704	.006	0.940
P-N2xcxs	.112	0.740	3.053	0.096
Platxcxs	2.085	0.158	.356	0.558
Pxcxs	.046	0.831	.947	0.343

F-statistics for multivariate test of equality of mean vectors (F-approximation of the liklihood ratio)= 2.553, D.F.=16 and 18. P<0.029.

Table 5a): showing the univariate and step down F values of variables following tone onset, and the corresponding p, and their interactions in the 'natural order'.

a multivariate population in which the variables of interest are independent. The anti-image test involves the inspection of the off-diagonal elements of the anti-image covariance matrix. In the absence of many zero off-diagonal elements, the indication is that the matrix is not appropriate for factor analysis. The third statistic indicates whether the variables belong together psychometrically (Dziuban and Shirkey 1974). The obtained indices of these measures for the data matrices input, particularly for the CNV data, suggested that the matrices were suitable for

B			C		
VARIABLE	STEP DOWN	P<	VARIABLE	STEP DOWN	P<
N1	3.011	0.092	P-N2	.129	0.721
Pxs	2.905	0.098	Plat	.074	0.787
P-N2xcxs	.046	0.832	P	4.486	0.042
N1xs	4.679	0.039	N1xc	.902	0.350
P-N2	3.272	0.081	P-N2xc	.324	0.574
Plat	.548	0.465	Platxc	.045	0.833
P	2.273	0.143	Pxc	4.189	0.051
N1xc	1.289	0.267	P-N2xs	.385	0.541
P-N2xc	3.982	0.057	Platxs	1.316	0.262
Platxc	.075	0.787	N1xcxs	.069	0.795
Pxc	3.500	0.074	Platxcxs	1.001	0.327
P-N2xs	5.211	0.032	Pxcxs	.168	0.686
Platxs	.486	0.493	N1xs	.757	0.394
N1xcxs	.216	0.647	P-N2xcxs	2.895	0.104
Platxcxs	.256	0.558	Pxs	10.492	0.004
Pxcxs	.947	0.343	N1	2.444	0.135

Tables 5 b) and c): showing step down F values for between group differences when variables of interest are placed at the begining, or at the end, of variables input to the analysis; univariate F values are unaffected (see Table 5a).

factoring, i.e. that the matrix was invertable, and therefore suitable also for multivariate analysis-of-variance.

Separate multivariance runs were completed for CNV data, and for ERP measures following light and tone onset, including and excluding main effects, comparing the differences between means for the two groups. Excluding main effects in the CNV data made no difference to the univariate or step down F values obtained, but significantly improved the multivariate F value. By contrast, the clearest results were obtained in the analysis of the ERP variables following tone onset including main effects; the analysis of which

VARIABLE	1	2	3	4	5	6
N1	.460	-.402	.155	-.600	-.265	-.111
P-N2	.601	-.615	-.239	-.269	.165	-.117
Plat	.273	.545	.039	-.034	-.201	-.536
P	.532	-.645	-.221	-.024	.290	-.103
N1xc	.639	.228	-.343	-.193	-.204	.241
P-N2xc	.664	-.217	.335	.281	-.084	.145
Platxc	.703	.132	.290	.136	.061	.252
Pxc	.276	-.330	.622	.471	.254	-.081
N1xs	-.221	-.515	.429	.440	-.156	-.265
P-N2xs	-.077	-.354	-.655	.335	-.440	-.143
Platxs	-.570	-.034	-.260	.013	.485	.264
Pxs	.240	.019	-.567	.564	-.311	.238
N1xcxs	-.214	-.612	-.468	-.026	.127	.074
P-N2xcxs	-.443	-.555	.250	-.242	-.345	.118
Platxcxs	-.852	-.139	-.083	.002	.008	-.216
Pxcxs	-.440	-.191	.521	-.127	-.365	.401

Table 5d): showing, reduced to three decimal places, the first six principle component loadings of the variables to be input to multivariate analysis-of-variance.

will, accordingly, be reported. No main or interaction effects, in either univariate or step down F values, were obtained in the multivariate analysis-of-variance of the ERP variables following light onset, and this analysis will not be reported further.

Table 4a) contains the summary statistics for the comparison of means between controls and reactive depressives for the CNV variables; only interaction effects being examined on this run. Against each interaction variable is shown the univariate and step down F values, which are computed automatically by the programme. The F approximation for the likelihood ratio is a measure of the extent to which the data are less likely to have arisen from a population described by the model when the null hypothesis is

A:CNV VARIABLES			B:VARIABLES TO TONE ONSET		
Natural order	Begining	End	Natural order	Begining	End
lat2xs (S)*		lat2xs (S)*	N1 (U+S)	N1 (U+S)	N1 (U)
			N1xs (U)*	N1xs (U+S)*	N1xs (U)*
amp2xcxs (S)*			Pxs (S)*	Pxs (S)	Pxs (S)*
lat2xcxs (S)	lat2xcxs (S)*		N2xcxs (S)		
latlxcxs (U)	latlxcxs (U+S)	latlxcxs (S)*		P-N2 (S)	
	ar2xc (S)*			P-N2xc (S)	
	arxs (S)*			Pxc (S)	pxc (S)*
		ar2xcxs (S)*		P-N2xs (S)*	
					P (S)*

Tables 6a) and b): Summary tables of vaiables, and their interactions, of interest on the separate multivariance runs. 'Natural order' denotes the initial order of variables input to multivariance, and 'begining' and 'end' indicate the placement of variables in subsequent runs. (U) and (S) indicate high univariate and step down F values respectively, and * a signif icant F value; c=condition, s=session.

true than when it is false. The smaller its value, the

higher is the validity in rejecting the null hypothesis. In this instance, of testing the difference between two sample means, the multivariance analysis-of-variance hypothesis of no difference between poulation means for the interactions just fails to reach significance ($F=1.964$, $p<0.086$). However, inspection of the tables produces other information that, with care, can be interpreted. The univariate F statistic depicted is exactly that which would have been obtained if only that single variable had been subjected to analysis-of-variance. The largest F value indicates the interaction having the largest between group difference relative to the within group variation, and vice versa. The step down F procedure partials out the between group effect in a single variable, or interaction, contributed to it from those preceding it in the order of variables in the analysis. Thus, the between group difference in all variables and their interactions , can be identified. However, it should be noted that the step down procedure is dependent on the order of the variables input to the analysis. In this instance, a three way interaction is indicated between latency to early peak amplitude and condition and session, which just fails to reach significance ($F=3.621$, $p<0.06$). Of the step down values, a significant two-way interaction, differentiating the two groups, is indicated between latency to late CNV peak and session ($F=5.394$, $p,0.03$) and a three-way interation between late CNV amplitude and condition and session ($F=6.522$, $p<0.02$). A three-way interaction effect between late CNV area and condition and session failed to reach the criterion acceptance level ($F=3.373$, $p<0.08$), as did that between early CNV amplitude and condition and session ($F=3.076$, $p<0.09$). No simple interaction effects were obtained with conditions for any of the variables. These interaction effects were subjected to further analysis by placing them at the begining and end of the variables to be tested (see Tables 4b) and c)) and included the three-way interaction for early amplitude with condition and session which, although presenting the most

significant univariate F, demonstrated an insignificant step down F value. Placing the variables both at the begining and end of the variable list for further analysis, which are then evaluated together is seen as the most conservative approach to the evaluation of results. Its effect is to more systematically partial out the between group variance that contributes to the within group for each variable of interest. As is apparent in these tables, and in the summary table 6a), the re-ordering of input variables can lead to the generation of other significant step down values.

Whilst a full interpreation of the obtained results will be presented in the discussion section, those of significance can be introduced here. Latency to early peak CNV amplitude, in interaction with either/or condition and session, produced group differences in the step down analysis. Latency to peak amplitude in interaction with condition and session produced a significant step down F value ($F=4.62$, $p<0.05$) when placed at the end of the list; that is after the removal of the between group variance contribution of all the other varaibles. Late peak CNV latency, in interaction condition and session, produced a significant difference between the groups (step down $F=4.80$, $p<0.03$), when place at the begining of the list, but not when placed at the end. Of the other variables producing interesting step down F values in the natural order of input of variables, late peak latency in interaction with session, produced a significant step down F when placed towards the end in the ordering of variables ($F=5.82$, $p<0.03$). However, other variables emerged with significant F values for the variable late area in interaction with condition ($F=7.69$, $p<0.01$) and with session ($F=5.88$, $p<0.02$) with the early ordered variable list. In the late reordering of variables significant step down F values were again obtained for late peak area in a three-way interaction with condition and session ($F=6.94$, $p<0.01$). This complex picture of interactions, and that of newly emerging significant F values

with the reordering of variables, can be explained by a complex correlation between these input variables. Such an explanation is partially corroborated by inspection of the principle component loadings (Table 4d)). It must, though, be stressed that over all variables taken together no significant group differences emerged. To investigate the possibility that group differences were obscured by intra-individual variance in the raw data, this was reanalised condensing ten data points and these new measures used as input to multivariate analysis-of-variance. No group differences, in either main or interaction effects emerged in this modified analysis.

The same approach to the analysis of the variables following tone onset was applied as to the CNV variables, described above. As indicated earlier, there are strong indications that the variable N1-P contains a marked contribution from the emitted positivity. Accordingly, this variable was transformed by the subtraction of the positive amplitude contribution, and the resultant interpreted as a pure measure of N1 amplitude, which was used as input to the multivariate analysis. These may be found in Tables 5a), b), and c), the corresponding principle components in Table 5d), and the overview in Table 6b).

Multivariate analysis-of-variance of the variables to tone onset produced significant group differences across all input variables taken together ($F=2.55$, $p<0.03$). Examination of the univariate statistic showed a non-significant main effect for N1 ($F=3.01$, $p<0.09$), but one which obtained significance in a two-way interaction with session ($F=4.40$, $p<0.04$); this disappeared in the step down analysis. Examination of the step down values revealed a highly significant F value for positivity in interaction with session ($F=12.60$, $p<0.001$) and a non-significant value for P-N2 in interaction with condition and session ($F=3.05$, $p<0.09$). These four variables were introduced at the begining and the end of the variable list for further multivariate analysis. N1 amplitude, as a main effect, demonstrated no increase in

significance when placed at the end of the variable list; indeed its step down significance level was slightly decreased. However, it continued to demonstrate a marked interaction effect with session, when placed early in the order of variables (univariate $F=4.40$, $p<0.04$, step down $F=4.68$, $p<0.04$), but not when placed at the end. The interaction between positivity and session was reduced in its step down significance level, when placed at the begining of the list of variables ($F=2.90$, $p<0.10$), but was highly significant when the between variable variance contribution was removed by placing it toward the of input variables ($F=10.49$, $p<0.004$). As with the CNV variables, a complicated inter-correlation between these input variables is suggested by the emergence of significant step down F values with the re-ordering of input variables. Thus a P-N1 interaction, which just failed to reach significance, with session (step down $F=3.98$, $p<0.06$) is evident with the early re-ordering of variables. With the late re-ordering of variables, a significant step down F value was obtained for positivity alone ($F=4.48$, $p,0.04$) and in interaction with condition ($F=4.20$, $p<0.05$). No significant results for P latency, either as a main or interaction effect, were obtained. Thus, in summary, it may be concluded that while main effects for N1 and peak positivity are indicated, these variables discriminated between groups most clearly in interaction with session.

DISCUSSION.

Attention, in the following discussion, will be directed, firstly, at results derived from the correlational studies and those evaluating group differences before turning to the more comprehensive multivariate application. Where relevent, previous studies will be included in the discussion of the various points introduced. The CNV, and ERPs to the single stimuli, will be discussed separately, in that order, before turning to more general points. The section will be concluded with an evaluation of the methodology, applied in the present study, together with proposals for future possible investigations.

Considering, firstly, the evaluation of the reliability of the CNV and ERP measures, certain introductory comments are necessary. Investigation of such emitted activity, as a possible correlate of psychopathologic disorder, requires that such measures themselves be reliable over time, as well as sensitive to experimental manipulations, in a control group. Previous evaluations of the test-retest reliability of the CNV have relied on correlational analysis of the waveform using short ISIs. Certain observations, already introduced above, may be summarised here. Correlational analysis evaluates the degree of association between two sets of scores and is insensitive to the absolute value of the measures. The Wilcoxon statistic was applied to determine the stability of individual scores within and between sessions. Evaluation of the stability of absolute measures clarifies the responsiveness of the measures themselves to the passage of time and to experimental manipulation. Besides providing a comparison group for reactive depressives, controls also permitted the basic evaluation of the reliability and sensitivity of the measures applied to describe the waveform using a long ISI. Whilst CNV activity in controls was recognisable in the graphs of the averaged waveforms, the separate components demonstrated differential

reliability over a period of months. A significant reliability coefficient was obtained only for early peak amplitude in the non-incentive condition. The measures themselves, however, were reduced over this period; this reduction reaching significance for the above measure and for the corresponding area and lengthening of early peak latency in the incentive condition. Variability, as a reliable measure of the CNV, is suggested by the significant correlations in the separate epochs between the non-incentive conditions and is reinforced by the absence of changes in central tendancy. The absence of such an association for the incentive condition reinforces the observation that controls responded idiosyncratically to the introduction of monetary incentives. Overall, a greater stability is suggested for the early part of the CNV over long periods of time. That, in the incentive condition, a significant reduction in central tendancy for early peak amplitude, and its latency, between sessions, without any corresponding significant correlations, suggests that in the second session controls produced qualitatively different responses to the incentive condition which altered the group structure. Thus, the early part of the CNV may be regarded as more reliable than the latter, which may reflect more exactly the degree of subjective participation and both between session and inter-individual changes in this involvement in the experimental task: an interpretation supported by the intra-session performance discussed below.

That high correlations are evident in session one for the area and amplitude, but not latency, measures suggests a short-term reliability. The concommitant, but non-significant, reductions in amplitudes, and increases in latencies, supports an interpretaion favouring non-systematic changes in responses on the part of subjects to the introduction of monetary incentives; which may, on a priori grounds, have been expected to increase in area and amplitude measures. The suggestion, above, that controls, in the second recording demonstrated a qualitatively different involvement in

the experimental task is supported by the lack of intra-session correlations and by the non-significant increase in area and amplitude. Although in neither session reaching significance, a motivational enhancement, reflecting the introduction of monetary incentives, is suggested only in session two. CNV variability produced no significant correlations in session one, but, in session two, variability in early and late epochs correlated between conditions; the former of these demonstrating a reduction.

Certain conclusions, in view of previous reports, may be offered at this stage. Reports of elevated baseline levels to the introduction of monetary incentives (McAdam and Seales 1969) or of increased late negativity to higher S2 significance (e.g. Gaillard 1977; Lang et al. 1978) are not supported in this study by the inter-session comparisons which suggest rather both non-significant increases and decreases in the area and amplitude measures used. Whilst it is not the present author's intention to join the debate over which adopted descriptor, be it 'motivation' or 'expectancy', best fits the psychological process involved, certain comments are in order. Neither motor preparation (Grünewald et al. 1979; Gaillard and Perdok 1979) as a determinant of late negativity, nor the facilitative effect of the 'O' wave on later responses (Loveless 1977) are supported by the present results in that, although average reaction time reduced between conditions, it did so independently of direction of change in the CNV measures. This is further supported by the absence of correlations between CNV measures and average reaction time. Similarly, the early CNV as a pure index of orienting (Loveless and Sanford 1973,1976; Peters 1977; Lang et al. 1978) is not confirmed, in that it reduces both within, and between, sessions; intra-session reductions might not be expected in the incentive condition where S1 contains more information or alerting characteristics. Such a variation may be introduced by a generalised motor readiness or organisation of behaviour (Picton et al. 1978; Simons et al. 1979; Picton 1980). The slow

supports the CNV as reflecting an intention to behave rather than performance processes per se (Näätänen 1978).

The test-retest reliability coefficients and stability indices obtained in this study do not support the CNV as composed of components capable of reflecting trait dispositional factors (Roth 1975). Whilst high inter-session retest reliability coefficients were obtained in session one, but not repoduced in session two, these tended to reduce in amplitude, albeit non-significantly, within sessions. Nonetheless, these may be taken as lending some support for the short-term reliability reported elsewhere (Roth et al. 1975; Tecce 1979; Timsit-Berthier et al. 1980); albeit that these researchers used much shorter ISIs. In the present study, the short-term reliability in session one may well reflect the lack of change in invovement between conditions, whereas, in session two, the possibility for significant correlations is vitiated by the tendancy to a non-significant increase in measures. However, over longer retest intervals, no support can be offered for the finding of Abraham et al. (1976) that mean acquisition amplitude was consistent on retesting, or for Roth's (1975) report of high reliability over weekly recording intervals, except for early CNV measures. The observed reduction in CNV measures, particularly early amplitude in the non-incentive condition and its corresponding area and latency in the incentive, qualifies the finding of Timsit-Berthier et al. (1980) of reduced pre-S2 amplitudes.

Turning to the reliability scores for reactive depressives first, significant test-retest correlation coefficients were obtained between sessions for late peak negativity and its corresponding area in the incenive condition and for latency to peak negativity in the non-incentive. Unlike controls between sessions, reactive depressives demonstrated increased amplitudes and shortening peak latencies, and so lending statistical support to those from visual inspection of the waveforms. These were significant, in the incentive condition, for late and total CNV area. This pattern of

significant correlations and shifts in central tendancy appears dissimilar to that displayed by controls; this reversal of pattern being also displayed in the within session comparisons. In session one, both early and late peak latency correlated significantly between conditions and, in session two, did late maximum amplitude. It is suggested that reactive depressives did not significantly and systematically respond to the introduction of monetary incentives in either session, as reflected in the absence of changes in central tendancy, excepting early peak amplitude latency in the second sesion. This is further reflected in the overlap between conditions in the waveforms of the second session. There is, further, the suggestion that, unlike normals, variability in CNV amplitude in the three epochs is not stable, in that significant test-retest coefficients were obtained only for early variability. The suggestion, above, of greater uniformity of responses in the second session, is supported by significant correlations for all three epoch measures of variability between conditions, and for a significant correlation only for early variability within session one. This latter supports the previous absence of correlations between conditions suggesting an idiosyncratic response on the part of reactive depressives to the monetary incentive condition in session one, and indicate, further, that the responses that any individual produced fell in the same range under both conditions.

The suggestion, that CNV amplitude variability may be an useful index of CNV activity, is supported by reference to the between condition comparison of session one for endogenous depressives. Whilst both positive and inverse correlations between conditions in the CNV measures were obtained, variability in the three epochs correlated significantly. No systematic changes in central tendancy were observed. However, the inverse correlation in early peak latency is difficult to explain. Clearly, in view of the group size, results dealing with endogenous depressives should not be exaggerated.

To conclude, certain comments may be made concerning previous reports on test-retest reliability in psychiatric groups. The examination of group means and visual inspection of the waveforms indicates an increase in late peak amplitude and, possibly, area and a slight reduction in early, which is substantiated by the Wilcoxon analysis. Combining correlational and central tendancy results indicates that reactive depressives uniformly, so as to maintain their position within the group, increased negativity which proved significant for the early and total area measures. These results make more specific those of McCallum (1969) reporting a non-significant increases over a period of 1-2 months, and of Knott (1976) of increases in negativity of depressives during hospitalisation.

No differences for the CNV measures, or their variability, in either session, in non-incentive conditions, were revealed by the application of Mann-Whitney analysis between reactive depressives and controls. In the non-incentive condition of session one, differences between endogenous depressives and controls were obtained only for variability in CNV amplitude in the early epoch; latency to peak amplitude, in the non-incentive, and early peak amplitude in the incentive, both failed to obtain acceptable significance levels. This group difference for the early CNV epoch variability was apparent also between reactive and endogenous depressives. In both instances, endogenous depressives demonstrated lower variability: this may be attributable to their smaller group size. Whilst reactive depressives demonstrate both lower early and late amplitudes, these differences are non-significant. Although non-significant also, reactive depressives in session one demonstrate greater late and total CNV areas. Thus, the assertion that psychiatric patients are more prone to CNV reductions (McCallum 1969; Cohen 1974; Roth 1975) is not substantiated. Rather, weight is added to the proposition that, with short ISIs, CNV processes may overlap, without having permitted a full morphological development. These findings lend support

to those of Giedke and Bolz (1980) of no differences between primary depressives and controls in a R.T. task, and extend them through the inclusion of monetary incentives as not producing group differences. Although more fully discussed below, no support is found for their report that high depression scores, as here measured on the Beck .scale, is associated with small CNV area. In this context, the suggestion of Dongier et al. (1977), that those suffering 'characterological' depression have developed homeostatic adjustments, so that no electrical sign of cerebral dysfunction remains apparent, is well taken. Similarly, the results do not contradict a spontaneous recovery of neurotransmitter level or synthesis, which is of relevence to the hypothesised monamine theory of depression.

The report of Timsit-Berthier et al. (1973), of significantly lower maximum negativity in manic-depressives, is not substantiated in this study. Nor are the smaller CNVs in psychotics, reported by Roth (1975), or in both manic and depresive episodes of affective psychosis (Shagass 1979b). In part this may be due to the shorter ISIs used in the former studies, and in part by the small sample size in this. However, it may be worth noting, in passing at this stage, that Beck scores significantly distinguished between controls and both reactive and endogenous depressives, and between the depressive groups.

As mentioned in the presentation of results on CNV variable inter-recording relationships and group differences, and highlighted in the multivariate analytic demonstration of the absence of significant main or interaction effects, CNV variables present a complex mosaic of interaction, and can be applied in no simple mannner to a description of the two groups involved. The absence of main effects itself further refutes the assertion of Giedke and Bolz (1980), that a reduced CNV area is a constant sign of depressive illness. Partialling out between group variance from that within, the two way interaction between latency to late peak amplitude and session permits controls and react-

ive depressives to be significantly discriminated. This seems to indicate that the lengthening of late peak latency for controls, most apparent in the incentive condition between sessions, and its shortening for reactive depressives is, after the removal of condition effects, significant. Excepting the non-incentive condition of session one, reactive depressives as a group, demonstrate earlier late peak latencies which, although not significant under the separate Mann-Whitney analyses, emerge as significant under multivariate. Three-way interaction effects are particularly difficult to interpret. However, of special interest here, is that suggested between late CNV area and condition and session; supporting that introduced above in the discussion of correlational and central tendancy results. What may be signified here is the increase in late area demonstrated by depressives in the incentive condition between sessions, but not in the non-incentive, and the suggestion of an overall reduction in the measure demonstrated by controls. Thus, depressives are discriminable from controls in their elevated areas to the introduction of incentives after a period of hospitalisation.

In conclusion, the results derived in the present study pertaining to the CNV support a polymorphous interpretation of its component processes. It has been asserted that ERP components are modifiable by the subject's prior experience, decisions and expectations as modulated by the nature, context, and demands of the current task. Insofar as the CNV components reflect the application of such subjective factors to performance, no isomorphic relationship is supported. Nonetheless, the CNV is a reliable electrocortical manifestation of involvement in experimental tasks per se. This dilemma may, perhaps, be resolved by accepting the CNV as indicating the regulation of general arousal processes to perform (Simons et al. 1978; Picton 1980). Such would be responsive to, for example, involvement in experimentation per se, but need not interact monotonically with any specific experimental variable. Such would rationalise, for

example, the lack of group differences, or the lack of significant amplitude or area elevation to monetary incentives on the part of normals. Applying the cognitive psychological terminology of Cooper et al. (1978), such would suggest that depressives operated more in the 'scopeutic' mode and controls the 'categoric'. Knott's (1972) assertion that slow potential activity needs to be understood in the terms of interacting psychological variables is supported here in the demonstration of group differences only in terms of interaction effects in the step down F values.

Turning attention next to the elicited potentials to tones, and firstly to the status of 'P'. An unequivocal solution to the identity of this component is negated by the recording from only the vertex in this experiment so that it is uncertain whether it would appear frontally or more posteriorly dominant. Its latency, nonetheless, corresponds well with that of the P3a at approxiamately 240 msec (range:220-280 msec) (N.Squires et al. 1975), but a P300, with a latency of 220 msec, is also reported by Roth and Cannon (1972) and at 200-300 msec (Smith et al. 1070). The P3a has been elicited while subjects engage in another task (Tueting 1978), by shifts in stimulus intensity or loudness (N.Squires et al. 1975) whether these are attended or unattended (Picton et al. 1978), and in missing stimulus paradigms (Sutton et al. 1967). Vaughan et al. (1980) identified a positive component, of 245 msec latency, to stimuli following a rare stimulus and which required a response. In the ensuing discussion 'P' will be termed 'P240', the characteristics of which are reviewed below.

Thus, whilst the latency of the P240 component suggest that it belongs to the family of P300s, these latter are termed significant in reflecting as much the subject's cognitive approach to sensory information processing as any psychological principle. Without preempting the ensuing discussion on the sensitivity of the P240 to changes in experimental conditions, and the ability of such to reflect

group differences, certain introductory remarks are in order to satisfy, from a neuropsychological viewpoint, the inclusion of the P240 among P300 phenomena. The P300 follows a point in time at which relevent information becomes available, even when such an occurence is predictable. Thus, strongly held hypotheses, which are confirmed, can elicit large P300 waves. Such activity may appear relatively early, as indicated by the study of Vaughan et al. (1980) mentioned above. However, in the present study, the warned tone required no immediate response, but rather indicated the begining of the reaction time task foreperiod. Further, Duncan-Johnson and Donchin (1978) reported that ERPs were greatly reduced when a warning stimulus indicated the a priori probability of the coming stimulus: in the present study, this probability was one. The elicited P240 activity, although in a different sensory modality to the P300 elicited to lights, was of markedly higher amplitude. Accordingly, probability as such cannot be invoked here as the sole determinant of the P300. Rather other processes need to be involved. Tone presentation gains additional importance by signalling an interval at the end of which a speedy response would be required. P240 activity may, therefore, index subjective involvement, i.e. attention to the stimuli, in a task in which a controlled motor response is mandatory. As such, it would be similar to the explanatory model in terms of contingent probability posited by Cambell et al. (1979). The P2, of earlier latency and bound to stimulus characteristics, is not considered a viable descriptor for the P240 recorded here. In summary, the P240, obtained in this study, may safely be accepted as influenced by engagement in the experimental task comparable to that of the P300 family. Further comments on its nature will be offered at the conclusion of the ensuing discussion on its responsiveness to experimental manipulation and ability, thereby, to discriminate between controls and reactive depressives.

Very high test-retest correlation coefficients to the

amplitude and latency measures of the ERPs to tones, including N1 alone and compounded with P240, were obtained for controls between sessions in both conditions. Of the measures, P240 amplitude was significantly elevated in the incentive condition of the second session, but no other measure demonstrated significant fluctuation in values. These measures, also, all demonstrated very high intra-session correlation coefficients and, interestingly, changeability in the absolute values. N1-P240, P240-N2 and N1 were significantly reduced between conditions in both sessions, as was P240 amplitude also in session two. These results support, firstly, the, at least patial, independence of N1 and P240, on the basis of independence of condition and session effects, and secondly, support the inclusion of the P240 within the class of event-related positive potentials.

Reactive depressives presented significant correlations between sessions for P240 amplitude and latency, and for N1 amplitude, in both conditions. Apart from a reduction in mean amplitude for N1 in the incentive conditions between sessions, no significant shifts in central tendancy were observed. All of these measures demonstrated high within session, i.e. between condition, correlations. Significant reductions, indicating the systematic reduction in absolute values, by reactive depressives as a group, for the amplitudes of N1-P240, P240-N2, and P240 in session one, and to N1-P240 amplitude alone in session two, were observed. Accepting that it is often difficult to separate effects of N1 and late positivity, these results parallel those of controls remarkably well. That these may respond independently is suggested by the results from endogenous depressives, who demonstrate, within session one, between condition correlations in N1-P240 and P240 amplitudes; with significant reductions observable in the former amplitude and in that of N1.

Group comparisons, on the basis of ERP measures to tones, [illegible] a significant difference between controls

and reactive depressives only for N1 amplitude in the incentive condition; it being higher for the latter group. Comparison of endogenous depressives and controls produced significantly higher N1-P240 amplitude in both conditions, and N1 in the non-incentive, for the former group. These also demonstrated a shorter latency to maximum P240 amplitude in the non-incentive condition. This measure also demonstrated a significantly shorter latency than that of reactive depressives in this condition, but no other group differences obtained.

These findings do not support those of Levit et al. (1973) or of Shagass et al. (1978) reporting no differences in N1 amplitude between neurotic depressives and controls, but lend support to those of Giedke et al. (1980) of N1-P2 reduction in depressives; these latter authors did not report N1 amplitude alone. However, the non-significant P240 amplitude differences, on the basis of Mann-Whitney analysis, are in agreement with Levit et al. (1973) and Shagass et al. (1978). Latency to P240 peak in this study discriminated between endogenous and reactive depressives, in the non-incentive condition, but not depressives from normals. Although in agreement with Levit et al. (1973) and Shagass et al. (1978), that P240 amplitude alone demonstrated non-significant differences, N1 amplitude, either alone or in conjunction with P240 positivity, did separate endogenous depressives from controls.

The application of multivariate analysis, however, demonstrated significant group differences between controls and reactive depressives when only the unique variance in any variable remained, i.e. after the removal of variance to each variable from other variables. Controls overall demonstrated higher amplitude P240, but it is to the interaction of this variable with condition and session separately that attention should be focused. Within both sessions, depressives apparently reduced this amplitude to a greater extent than normals with the introduction of incentives. Across sessions, and particularly in the non-incentive condition,

depressives increased P240 amplitude to a greater degree than normals. This interpretation, whilst highlighting the need to examine variables in their psychological setting, i.e. in interacting with manipulation of experimental variables, does not confirm the absence of group differences in late positivity previously reported (Levit et al. 1973; Shagass et al. 1978). Examination of conditions within sessions separately does not allow these differences to emerge. Further, whilst in this analysis, differences in the main N1 effect which just fail to reach significance are obtained, it is in the interaction with session that group differences are apparent; both in univariate and step down F values. The suggestion is that, in both conditions over sessions, depressives reduced N1 amplitude to a much greater extent than controls. This change has to be viewed independently of changes within P240 amplitude. P240 latency, either alone or in interaction with condition or session, produced no group differences. However, fuller consideration of these measures is postponed until the conclusion of the discussion of elicited responses to light onset, introduced below.

Controls demonstrated both high inter-, and intra-, session correlations in all ERP measures to light onset. With the exception of P300 latency in the non-incentive condition, all measures were stable between sessions. Of interest, significant reductions in P300 amplitude were demonstrated, in both sessions, to the introduction of the incentive condition; in which these stimuli might have been expected to contain higher significance. This reduction may account for that also seen in session one in N1-P300 and P300-N2 amplitudes, in that no significant changes in N1 amplitude alone were observed. With the exception of latency measures, a remarkably similar picture is demonstrated by reactive depressives. In neither session did the slight reduction in amplitudes and shortening of latencies between conditions reach significance. Further, no measure produced any significant group difference which, with the

exception of P300 amplitude in the second session, were reduced in reactive depressives.

Whilst these results are in close agreement with those of Levit et al. (1973) and Shagass et al. (1978) concerning the lack of psychopathological contrasts, discussed above, they are, in the light of responses to tones, anomalous. The waveform elicited to lights replicates the latency and amplitude, typical of the P300, with greater fidelity than that to the warning tone. The clear indication is that all subjects did not regard light onset as the critical stimulus; rather the first tone determined the begining of involvement in the task. Such a contention is supported in that both N1 and P240 amplitude, alone or in interaction with condition or session, demonstrate changes associated with changes in information processing demands, and that the P240 was reduced in amplitude in the incentive condition in both sessions. Thus, although the timing of the light delivery was uncertain, its occurence delivered minimal information which was, more importantly, contained in the tone signalling the begining of the reaction time task. Nonetheless, the lack of group differences in the former indicate a comparable degree of involvement to a non-critical stimulus.

One further issue may reasonably be addressed at this stage, and that concerning the appropriate psychological descriptor for the processes involved in the ERP generation to tones. Whilst no absolute answer may be given, arousal processes alone are not considered capable of explaining changes in P240. Both differential preparatory arousal and the reactive change following stimulation would require monotonic changes in P240 amplitude. As demonstrated, this reduces within sessions, but increases over time. Further, the reactive change hypothesis is weakened in that only minimal negativity was observed pre-tone for both groups which were, nonetheless discriminable in terms of P240 and N1 interaction effects. Insofar as arousal processes are implicated in depression, reflected in environmental react-

ivity, depressives might be expected to exhibit larger P240. As demonstrated, the reverse is apparent.

Turning, finally, to a brief consideration of the relationship between the behavioural and electrocortical data. No consistent relationship was observed between any the CNV or ERP variables and average reaction time which, also, alone did not discriminate between the groups. Instead a common responsiveness to experimental manipulation was observed. Group differences were apparent in both sessions in the Bfs but, as indicated earlier, no obvious relationship exists between this measure and either the cnv and ERP measures or with average reaction time. Similarly, the Beck scale demonstrated no systematic relationship with CNV or ERP variables. The positive association of Beck score and early and total CNV area does not support the inverse relationship reported by Giedke and Bolz (1980). The absence of correlations for depressives in the incentive condition of session one, and at all in session two, suggests that they modified their performance, as reflected in the CNV, independent of any depression rating. The positive correlation in the non-incentive condition of session one suggests that those with the highest scores, including controls, were the most involved, indicating arousal, by experimentation per se; this being reflected primarily in the measures of the early CNV waveform.

The lack of distinct group differences make psychopathological evaluation difficult. As noted in the introduction, the identification of functional impairment with specific deviations in evoked potentials from those determined as normal are suspect on, at least, theoretical grounds; an evaluation consolidated by the failure to produce experimentally reproducable findings. Psychomotor retardation, itself contradicted by the shorter reaction times obtained in this study, might be demonstrated by reactive depressives in delayed peak latencies in the CNV and ERPs. None is supported by reference to ERPs to the lights and tones, and whilst group differences are, equivoc-

ally, suggested in late peak CNV latency in interaction with condition and session, it appears that reactive depressives presented an earlier peak latency than controls, and one that reduced across both conditions and sessions. Thus, for psychomotor retardation to have electrocortical correlates, these must operate at a later stage, e.g. one inhibiting response: such is not supported in the data. It further demonstrates that depressives can be motivated; the comment by Roth (1975) on patient's over-cooperativeness may be apposite here. Turning, finally, to the concept over reactivity to environmental events and the N1 and P240 components to tone onset. The higher N1 amplitude demonstrated by reactive depressives in session one, significant under Mann-Whitney analysis, and its interaction with session, indicating a significant reduction under multivariate analysis, strongly suggests a higher responsiveness to stimulation, which reduced over time. This is not in accord with Levit et al. (1973), who concluded that N1 amplitude contributed little, if anything, to N1-P300 group differences. These differences also argue against the elevated baseline negativity, posited by Roth (1975), in depression and against pre-tone baseline deviation as a source of artifact, as discussed in the result section, in this experiment. Whilst environmental reactivity may be supported by lower P240 amplitude throughout, in that elevated stimulus-bound processing in session one may inhibit this later processing, that this latter component may reflect other aspects of subjective involvement is indicated by the effects of both condition and session. However, in session two, the markedly reduced N1 amplitude is accompanied by only marginal increases in P240 amplitude in both conditions. Such requires the rejection of N1 and late positivity as demarkating exclusively stimulus and response modes of attention respectively.

In conclusion, certain observations are advanced in the light of the present experience, and developments in the field, from which future research efforts might profit.

Firstly, multichannel recording is recommended along the midline and over both hemisheres in the greater topographical precision, and hense application to neuropsychological interpretation, of event-related potentials that it offers. Such also facilitates, secondly, multivariate data analysis by increasing the number of sources of information which, although containing linearly dependent data, should display, at most limited, inter-electrode interaction. The application of principal component analysis to the covariance matrix, using the varimax rotation method, or time series analysis, would bring additional power to the resolution of unique and common electrocortical processes. Thirdly, as suggested in the reporting above, the control group presents suprising, if not atypical, responses. Greater control is recommended to obtain a naive group to the concept of depression and the process of neuropsychological recording, and one that has more time available to participate in experimentation. Forthly, although the fixed order of conditions in this experiment is not seen to have distorted inter-group comparisons or to have exerted a systematic effect on the variables, such a procedure should be avoided. Motivation, as a heuristic device, often defies experimental validation and, accordingly, it is questionable as to whether the experimental paradigm, and especially in the incentive condition, was similarly construed by the two groups participating. Thus, changes in the CNV and ERP measures may, at best, be regarded as corollaries of subjective involvement in an, albeit artificial, immediate environment into which other psychological dynamics could intrude. Finally, what, in all probability, further investigation of depression could demonstrate is not that qualitatively distinct aspects of the ERP waveform for depression can be identified, but rather that such measures exist on a continuum on which, often small, quantitative differences from normal must be evaluated in an overall, and interactive, context.

Appendix A1): the translated Edinborough Handidness Inventory.

Hatten Sie jemals die Tendenz zur Linkshändigkeit?

Bitte ankreuzen: Ja Nein

Geben Sie bitte mit "+" in der entsprechenden Spalte an, welche Hand Sie für die folgenden Aktivitäten bevorzugen. Wenn eine Aktivität von Ihnen mit einer der beiden Hände durchgeführt wird, kreuzen Sie bitte mit "+" an. Wenn Sie sich nicht entscheiden können, kreuzen Sie beide Spalten an.

Einige der Aktivitäten werden mit beiden Händen durchgeführt. In diesen Fällen ist der Teil der Aktivität oder des Gegenstandes in Klammern angegeben, für die die Handbewegung erfragt wird.

Versuchen Sie, möglichst alle Fragen zu beantworten und lassen Sie möglichst nur die aus, bei denen Sie keine Erfahrung mit der Aktivität oder dem Gegenstand haben.

	R	L
1. Schreiben		
2. Zeichnen		
3. Werfen		
4. Schere		
5. Zahnbürste		
6. Messer (ohne Gabel)		
7. Löffel		
8. Kehren (obere Hand am Besenstiel)		
9. Streichholz anzünden (Streichholz)		
10. Schachtel öffnen (Deckel)		
11. Mit welchem Fuß treten Sie bevorzugt einen Ball?		
12. Mit welchem Auge schauen Sie durch den Sucher eines Fotoapparates?		

Appendix A2): the post-experimental performance self-evaluation sheet.

Sie hätten heute maximal DM 10.- gewinnen können.
Wieviel bedeutet Ihnen dieser Betrag?
Kreuzen Sie bitte auf der folgenden Skala die Antwort an.

Ein Gesamtgewinn von DM 10.- hätte heute für mich bedeutet

sehr wenig — sehr viel

Wie zufrieden sind Sie mit Ihrer heutigen Gewinnleistung?

sehr unzufrieden — sehr zufrieden

Appendix A3). the Beck Depression Questionaire.

F r a g e b o g e n Beck

Bitte kreuzen Sie zu jedem Groß-Buchstaben, also von A bis T jeweils den Satz an, der Ihre Stimmung, Ihr Gefühl am zutreffendsten beschreibt.

A ( ) Ich bin nicht traurig.

( ) Es ist mir verleidet, oder: Ich bin traurig.

( ) Ständig ist es mir verleidet, oder: Ich bin immer traurig und kann nichts dagegen tun.

( ) Ich bin so traurig oder unglücklich, daß es mir weh tut.

( ) Ich bin so traurig oder unglücklich, daß ich es nicht mehr aushalten kann.

B ( ) Ich bin nicht besonders pessimistisch oder entmutigt in bezug auf meine Zukunft.

( ) Ich fühle mich mutlos in bezug auf meine Zukunft.

( ) Ich glaube, daß ich nichts zu erhoffen habe.

( ) Ich glaube, daß ich nie mehr aus meinen Sorgen herauskomme.

( ) Ich habe den Eindruck, daß meine Zukunft hoffnungslos aussieht und daß sich meine Lage nicht bessern wird.

C ( ) Ich habe nicht das Gefühl, versagt zu haben.

( ) Ich habe das Gefühl, mehr versagt zu haben als andere Leute.

( ) Ich glaube, ich habe wenig zustande gebracht, was einen Sinn oder Wert hat.

( ) Wenn ich mein Leben betrachte, sehe ich nichts als Versager.

( ) Ich glaube, ich habe komplett versagt.

D ( ) Ich bin nicht besonders unzufrieden.

( ) Ich langweile mich meistens.

( ) Ich habe an nichts mehr dieselbe Freude wie früher.

( ) Überhaupt nichts mehr verschafft mir Befriedigung.

( ) Ich bin mit allem unzufrieden.

E ( ) Ich fühle mich nicht besonders schuldig.
( ) Ich empfinde mich sehr oft als schuldig oder wertlos.
( ) Ich fühle mich schuldig.
( ) Ich empfinde mich jetzt praktisch immer als schlecht oder wertlos.
( ) Ich betrachte mich als sehr schlecht oder wertlos.

F ( ) Ich habe nicht den Eindruck, eine Strafe zu verdienen.
( ) Ich glaube, daß mir etwas Schlimmes passieren könnte.
( ) Ich habe den Eindruck, daß ich jetzt oder bald bestraft werde.
( ) Ich glaube, daß ich es verdiene, bestraft zu werden.
( ) Ich will bestraft werden.

G ( ) Ich bin nicht enttäuscht von mir.
( ) Ich bin enttäuscht von mir.
( ) Ich habe mich nicht gern.
( ) Ich kann mich nicht ausstehen.
( ) Ich hasse mich.

H ( ) Ich habe nicht den Eindruck, schlechter als andere Leute zu sein.
( ) Ich achte sehr gut auf meine eigenen Irrtümer oder Fehler.
( ) Ich mache mir Vorwürfe über alles, was nicht klappt.
( ) Ich habe den Eindruck, viele schlimme Fehler zu haben.

I ( ) Ich denke nicht daran, mir etwas anzutun.
( ) Ich denke manchmal daran, mir etwas anzutun - aber ich werde es nicht tun.
( ) Ich glaube, ich wäre besser tot.
( ) Ich habe genaue Pläne, Selbstmord zu machen.
( ) Ich glaube, für meine Familie wäre es besser, wenn ich tot wäre.
( ) Ich würde mich töten, wenn ich es könnte.

J ( ) Ich weine nicht mehr als gewöhnlich.
( ) Ich weine häufiger als gewöhnlich.
( ) Ich weine jetzt die ganze Zeit und kann nicht aufhören damit.
( ) Auch wenn ich es möchte, kann ich jetzt nicht mehr so weinen, wie ich es früher tat.

K ( ) Ich bin nicht gereizter als sonst.

( ) Ich bin rascher verärgert (oder gereizt) als gewöhnlich.

( ) Ich bin ständig gereizt.

( ) Alles, was mich gewöhnlich geärgert hat, berührt mich nicht mehr.

L ( ) Ich habe das Interesse für andere Leute nicht verloren.

( ) Ich interessiere mich weniger als gewöhnlich für andere Leute.

( ) Ich habe fast alles Interesse für andere Leute verloren und habe wenig Mitgefühl für sie.

( ) Ich habe alles Interesse für andere Leute verloren, sie sind mir total gleichgültig.

M ( ) Ich kann mich so leicht entscheiden wie gewöhnlich.

( ) Ich bin jetzt weniger selbstsicher und versuche, Entscheidungen aus dem Weg zu gehen.

( ) Ich kann keine Entscheidungen mehr treffen, ohne daß man mir dabei hilft.

( ) Ich kann überhaupt keine Entscheidungen mehr treffen.

N ( ) Ich habe nicht das Gefühl, schlechter auszusehen als gewöhnlich.

( ) Ich befürchte, daß ich gealtert oder unvorteilhaft aussehe.

( ) Ich habe das Gefühl, daß ich zunehmend schlechter und unvorteilhafter aussehe.

( ) Ich habe den Eindruck, häßlich und abstoßend zu wirken.

O ( ) Ich arbeite so leicht wie vorher.

( ) Es braucht eine zusätzliche Anstrengung, an etwas heranzugehen.

( ) Ich arbeite nicht mehr so gut wie gewohnt.

( ) Ich muß mir sehr große Mühe geben, um etwas zu unternehmen.

( ) Ich bin unfähig, auch nur die kleinste Arbeit zu verrichten.

P ( ) Ich schlafe so gut wie gewöhnlich.

( ) Am Morgen wache ich müder auf als sonst.

( ) Ich wache eine bis zwei Stunden früher auf als sonst, und ich habe dann Mühe, wieder einzuschlafen

( ) Ich wache jeden Morgen sehr früh auf, und ich kann nicht mehr als fünf Stunden schlafen.

Q ( ) Ich werde nicht rascher müde als gewöhnlich.

( ) Ich werde rascher müde als gewöhnlich.

( ) Alles, was ich tue, macht mich müde.

( ) Ich fühle mich zu müde, um irgend etwas zu tun.

R ( ) Mein Appetit ist nicht schlechter als gewöhnlich.

( ) Mein Appetit ist nicht so gut wie gewöhnlich.

( ) Mein Appetit ist jetzt viel schlechter.

( ) Ich habe überhaupt keinen Appetit mehr.

S ( ) Ich habe nicht an Gewicht verloren - oder, wenn ich abgenommen habe, seit ganz kurzer Zeit.

( ) Ich habe mehr als 2 Kilo abgenommen.

( ) Ich habe mehr als 4 Kilo abgenommen.

( ) Ich habe mehr als 7 Kilo abgenommen.

T ( ) Ich habe keine kürzliche Veränderung in meinen sexuellen [illegible] bemerkt.

( ) Ich interessiere mich weniger als gewöhnlich für Sexuelles.

( ) Ich interessiere mich viel weniger für Sexuelles.

( ) Ich habe jedes Interesse für Sexuelles verloren.

U ( ) Meine Gesundheit beschäftigt mich nicht mehr als gewöhnlich.

( ) Ich bin sehr mit Unwohlsein und Schmerzen beschäftigt, z.B. mit Bauchweh oder Verstopfung, oder mit anderen unangenehmen körperlichen Empfindungen.

( ) Meine körperlichen Empfindungen beunruhigen mich so sehr, daß es mir schwierig wird, an etwas anderes zu denken.

( ) Ich bin von meinen körperlichen Empfindungen vollkommen in Anspruch genommen.

Appendix A4): the Befindlichkeitskala.

Inst _____ Stat _____ Datum _____ Wt _____

I-Nr _____ ICD-Nr _____ Uhrzeit _____ / _____ Tz _____

_____ V - IQ _____

Ab hier vom Patienten auszufüllen:

Name _____ Mädchenname _____

Vorname _____ Geburtsdatum _____ Alter _____ Jahre

Beruf _____ Geschlecht m/w

Im folgenden finden Sie eine Reihe von Eigenschaftspaaren. Bitte entscheiden Sie – ohne lange zu überlegen – welche der beiden Eigenschaften Ihrem augenblicklichen Zustand am ehesten entspricht. Machen Sie in das Kästchen hinter der eher zutreffenden Eigenschaft ein Kreuz. Nur wenn Sie sich gar nicht entscheiden können, machen Sie ein Kreuz in die Spalte „weder – noch". Lassen Sie keine Zeile aus.

Ich fühle mich jetzt:

		eher		eher	weder–noch
1.	frisch		matt		
2.	teilnahmslos		teilnahmsvoll		
3.	froh		schwermütig		
4.	erfolgreich		erfolglos		
5.	gereizt		friedlich		
6.	entschlußlos		entschlußfreudig		
7.	lustig		weinerlich		
8.	gutgelaunt		verstimmt		
9.	appetitlos		appetitfreudig		
10.	gesellig		zurückgezogen		
11.	minderwertig		vollwertig		
12.	entspannt		gespannt		
13.	glücklich		unglücklich		
14.	scheu		zugänglich		
15.	sündig		rein		
16.	sicher		bedroht		
17.	verlassen		umsorgt		
18.	ausgewogen		innerlich getrieben		
19.	selbstsicher		unsicher		
20.	elend		wohl		
21.	beweglich		starr		
22.	müde		ausgeruht		
23.	zögernd		bestimmt		
24.	ruhig		unruhig		
25.	schwunglos		schwungvoll		
26.	nutzlos		unentbehrlich		
27.	schwerfällig		lebhaft		
28.	überlegen		unterlegen		

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APPENDIX B.

The following contains the legend to the variable names included in graphical or statistical representations. Where variable name is followed by three digits, the last of these refers to the experimental condition, the middle to the recording session, and the first to the form of the variable, e.g. AMP111 refers to the early peak CNV amplitude in the non-incentive condition of the first session. Where only two digits are to be seen, these refer to session and condition in that order.

AMP1, 2	Early late peak CNV negativity
LAT1, 2	Latency to early late peak CNV negativity
AR1, 2	CNV area with the early late 2 second CNV epochs
TA	Total CNV area
EEG1, 2, 3	Average negativity variability in the early, middle, or late CNV epochs
TNP1	N1 to peak positivity following tones
TNP2	Peak positivity following tone onset to succeding peak negativity
TLAT	Latency to peak positivity
TMAX	Baseline to peak positivity following tone onset
TN	N1 negativity following tone onset
SDT	Standard deviation to maximum positivity following tones
LNP1	N1 to peak positivity following light onset
LNP2	Peak positivity following light to succeeding peak negativity
LLAT	Latency to peak positivity following light onset
LMAX	Baseline to peak positivity following light onset
LN	N1 negativity following light onset
SDL	Standard deviation of peak positivity following light onset
AVRT	Average reaction time
Bf	Befindlichkeitskala
Bk	Beck Depression Rating
ZUF	Self-rating scale

VARIABLE	CONT.	R.DEP.	E.DEP.	VARIABLE	CONT.	R.DEP.	E.DEP.
AMP111	10.01	8.50	7.94	AMP112	8.55	7.12	6.09
	(4.6)	(3.5)	(5.8)		(5.2)	(3.0)	(5.4)
AMP211	7.39	6.99	6.62	AMP212	7.32	5.80	6.30
	(5.1)	(4.0)	(6.0)		(5.7)	(3.0)	(4.4)
LAT111	1.39	1.34	1.32	LAT112	1.12	1.39	2.09
	(0.8)	(0.6)	(0.6)		(0.4)	(0.7)	(3.7)
LAT211	5.10	5.24	4.82	LAT212	5.16	5.04	4.76
	(0.7)	(0.6)	(0.6)		(0.7)	(0.7)	(0.7)
AR111	10.17	8.28	9.41	AR112	9.33	7.16	6.31
	(5.7)	(5.0)	(7.7)		(8.0)	(4.1)	(6.9)
AR211	8.13	7.84	8.45	AR212	8.15	5.87	9.85
	(7.1)	(6.5)	(9.7)		(8.2)	(4.9)	(12.3)
TA11	21.51	18.60	23.19	TA12	8.15	15.88	21.95
	(13.0)	(11.2)	(22.8)		(19.6)	(9.5)	(25.9)
TNP111	23.89	26.52	30.13	TNP112	19.51	23.23	22.89
	(14.3)	(8.7)	(9.1)		(10.9)	(7.0)	(5.2)
TNP211	22.17	21.16	21.64	TNP212	17.58	17.94	18.16
	(13.1)	(7.9)	(6.4)		(10.9)	(8.7)	(5.9)
TLAT11	244.4	253.3	222.8	TLAT12	242.8	244.8	237.1
	(29.5)	(41.1)	(13.8)		(24.7)	(40.7)	(29.3)
TMAX11	11.18	9.66	10.17	TMAX12	8.89	6.75	7.43
	(8.5)	(6.9)	(7.6)		(7.6)	(8.3)	(6.9)
TN11	12.04	15.17	19.96	TN12	10.05	14.83	15.46
	(7.7)	(7.4)	(4.2)		(5.1)	(8.3)	(4.3)
LNP111	20.06	19.19	16.77	LNP112	17.74	18.37	14.49
	(7.5)	(6.8)	(3.3)		(6.0)	(7.2)	(3.5)
LNP211	19.74	17.76	14.49	LNP212	17.25	16.18	12.29
	(6.6)	(5.1)	(3.2)		(5.6)	(6.9)	(4.6)
LLAT11	355.6	347.4	360.0	LLAT12	342.2	344.2	368.6
	(47.8)	(46.3)	(41.6)		(42.2)	(41.9)	(67.2)
LMAX11	15.03	15.03	13.04	LMAX12	13.11	13.88	11.47
	(7.1)	(6.4)	(4.6)		(5.0)	(6.5)	(5.7)
LN11	4.77	3.96	3.73	LN12	4.38	4.26	3.01
	(4.1)	(5.0)	(4.7)		(3.5)	(3.8)	(2.9)

Appendix C1: showing the means and standard deviations for the CNV and ERP measures in session one for both conditions for controls, reactive depressives, and endogenous depressives. All amplitudes are shown in μv; CNV latencies in seconds, and ERP latencies in msec. (Appendix B contains the legend to variable names).

VARIABLE	CONTROL	REACT.DEP.	VARIABLE	CONTROL	REACT.DEP.
AMP121	7.40	7.63	AMP122	8.35	6.84
	(5.6)	(3.0)		(6.5)	(3.5)
AMP221	7.35	7.46	AMP222	7.62	7.96
	(4.1)	(3.7)		(4.5)	(5.5)
LAT121	1.68	1.67	LAT122	1.63	1.30
	(1.1)	(0.7)		(0.9)	(0.6)
LAT221	5.39	5.11	LAT222	5.19	5.07
	(0.5)	(0.6)		(0.6)	(0.6)
AR121	6.88	8.18	AR122	9.22	7.15
	(6.3)	(4.6)		(9.0)	(5.0)
AR221	7.26	9.67	AR222	9.15	9.82
	(6.0)	(5.6)		(5.7)	(8.5)
TA21	17.57	22.00	TA22	21.79	31.40
	(14.3)	(13.7)		(14.2)	(51.0)
TNP121	29.95	24.75	TNP122	27.45	20.49
	(10.8)	(7.4)		(11.7)	(5.6)
TNP221	25.44	20.89	TNP222	21.98	11.91
	(10.1)	(6.2)		(8.5)	(6.8)
TLAT21	233.8	246.2	TLAT22	256.9	251.1
	(20.6)	(37.0)		(42.3)	(44.0)
TMAX21	15.36	9.83	TMAX22	14.56	7.94
	(6.6)	(7.6)		(6.7)	(4.2)
TN21	11.78	11.93	TN22	9.76	11.29
	(7.5)	(8.4)		(7.3)	(5.9)
LNP121	18.75	16.25	LNP122	15.94	16.33
	(8.2)	(4.7)		(7.7)	(4.8)
LNP221	20.56	16.96	LNP222	17.47	15.79
	(6.9)	(4.9)		(6.1)	(4.2)
LLAT21	335.4	336.5	LLAT22	348.5	329.5
	(40.9)	(31.8)		(51.3)	(36.7)
LMAX21	12.91	14.26	LMAX22	10.22	12.69
	(6.4)	(3.9)		(6.1)	(4.4)
LN21	4.41	1.69	LN22	4.78	3.45
	(4.7)	(3.1)		(5.2)	(3.6)

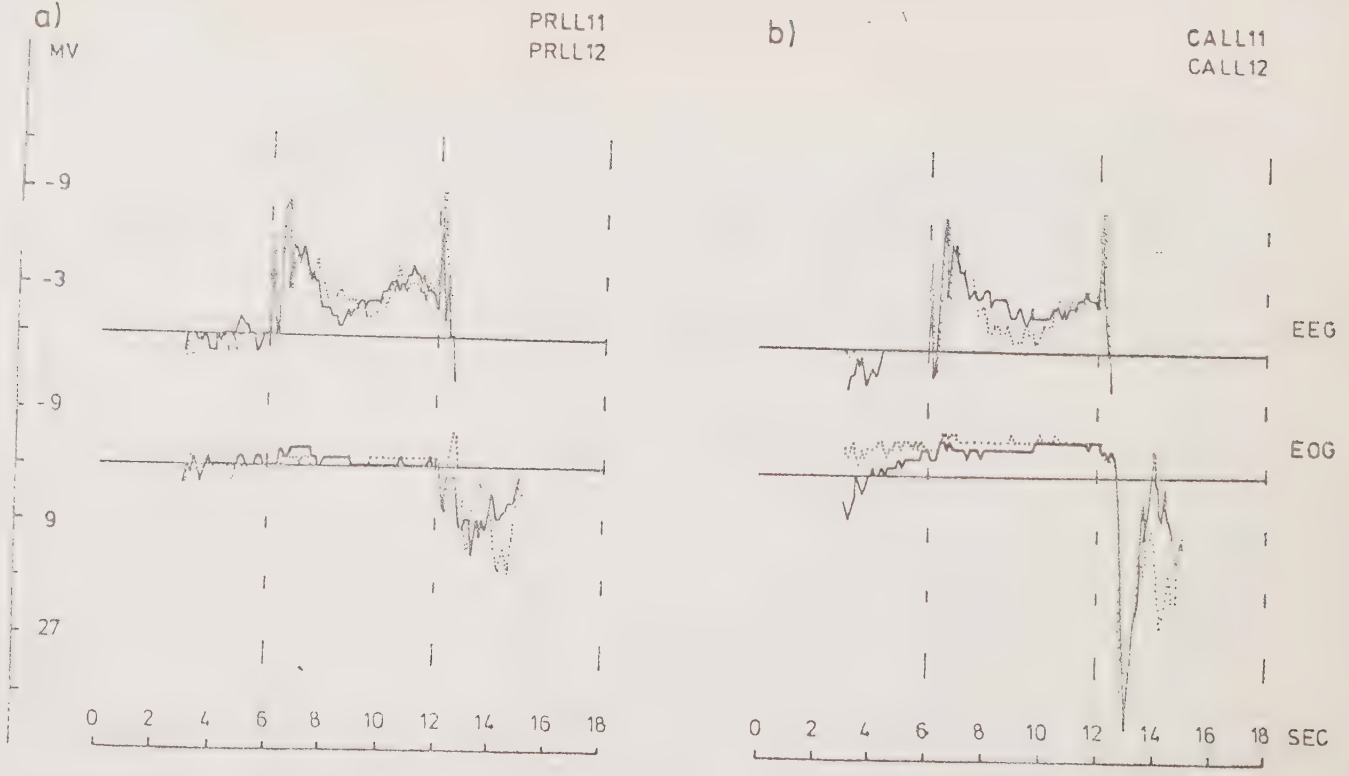
Appendix C2: showing the means and standard deviations for the CNV and ERP measures in session two for both conditions for controls and reactive depressives. All amplitudes are shown in μv, the CNV latencies in seconds, and ERP latencies in msec.

VARIABLE	CONT.	R.DEP.	E.DEP.	VARIABLE	CONT.	R.DEP.	E.DEP.
EEG111	11.48	11.88	8.47	EEG112	12.35	12.58	10.41
	(2.2)	(2.6)	(3.9)		(5.3)	(4.0)	(2.2)
EEG211	14.44	14.29	11.26	EEG212	13.75	14.43	11.42
	(3.9)	(3.3)	(2.3)		(5.0)	(3.5)	(2.1)
EEG311	14.79	14.64	11.55	EEG312	14.95	14.18	11.61
	(4.7)	(4.4)	(2.4)		(5.8)	(3.5)	(2.3)
SDT11	10.36	10.50	9.37	SDT12	11.00	10.89	9.70
	(3.3)	(2.9)	(2.8)		(3.1)	(3.1)	(4.0)
SDL11	12.06	14.25	10.57	SDL12	12.94	12.94	12.16
	(4.5)	(5.4)	(3.5)		(4.0)	(4.4)	(4.5)
EEG121	11.56	19.17		EEG122	10.83	11.79	
	(2.6)	(3.5)			(2.7)	(2.8)	
EEG221	13.45	12.79		EEG222	14.63	12.92	
	(3.8)	(2.5)			(7.9)	(2.9)	
EEG321	12.93	13.28		EEG322	13.77	13.36	
	(4.3)	(2.6)			(4.0)	(3.0)	
SDT21	10.97	10.78		SDT22	10.46	9.96	
	(2.5)	(3.3)			(3.1)	(2.4)	
SDL21	12.57	13.81		SDL22	10.16	12.04	
	(3.1)	(4.8)			(3.3)	(3.3)	

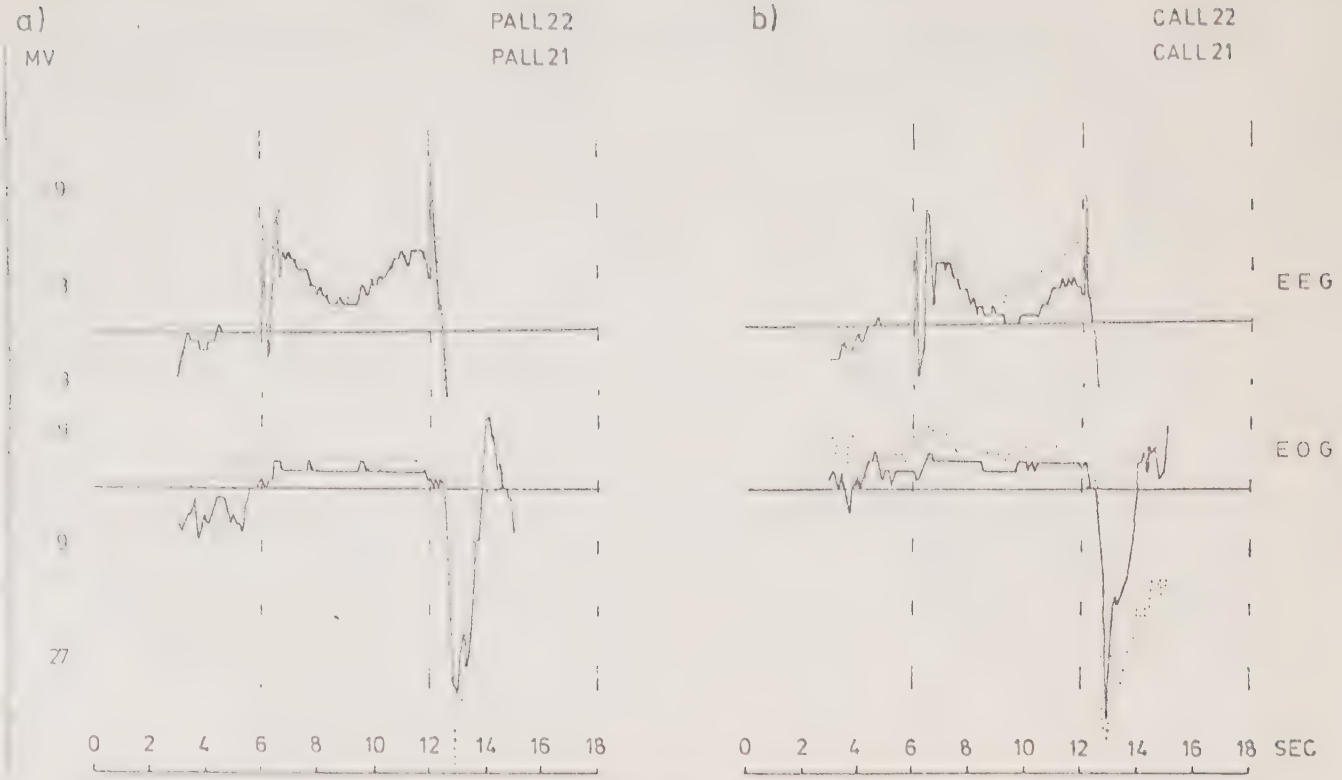
Appendix C3: showing the standard deviation around the individual raw data for the three CNV epochs, and the ERPs to light and tone onset, for normals, reactive and endogenous depressives in both conditions of both sessions.

VARIABLE	CONT.	R.DEP.	E.DEP.	VARIABLE	CONT.	R.DEP.	E.DEP.
AVRT11	303.8	274.6	269.5	AVRT12	278.2	238.2	245.6
	(84.9)	(84.2)	(147.3)		(72.6)	(54.5)	(122.2)
AVRT21	288.5	258.7		AVRT22	264.1	248.1	
	(63.9)	(74.5)			(52.9)	(59.1)	
Bf11	13.32	29.83	26.71	Bf21	12.22	29.19	
	(6.4)	(11.0)	(16.4)		(10.1)	(15.2)	
Bk11	2.42	29.00	19.20	Bk21		14.69	
	(2.5)	(9.2)	(6.8)			(12.35)	
ZUF11	66.00	67.00	57.3	ZUF21	55.56	60.47	
	(21.3)	(28.0)	(18.67)		(21.2)	(25.0)	

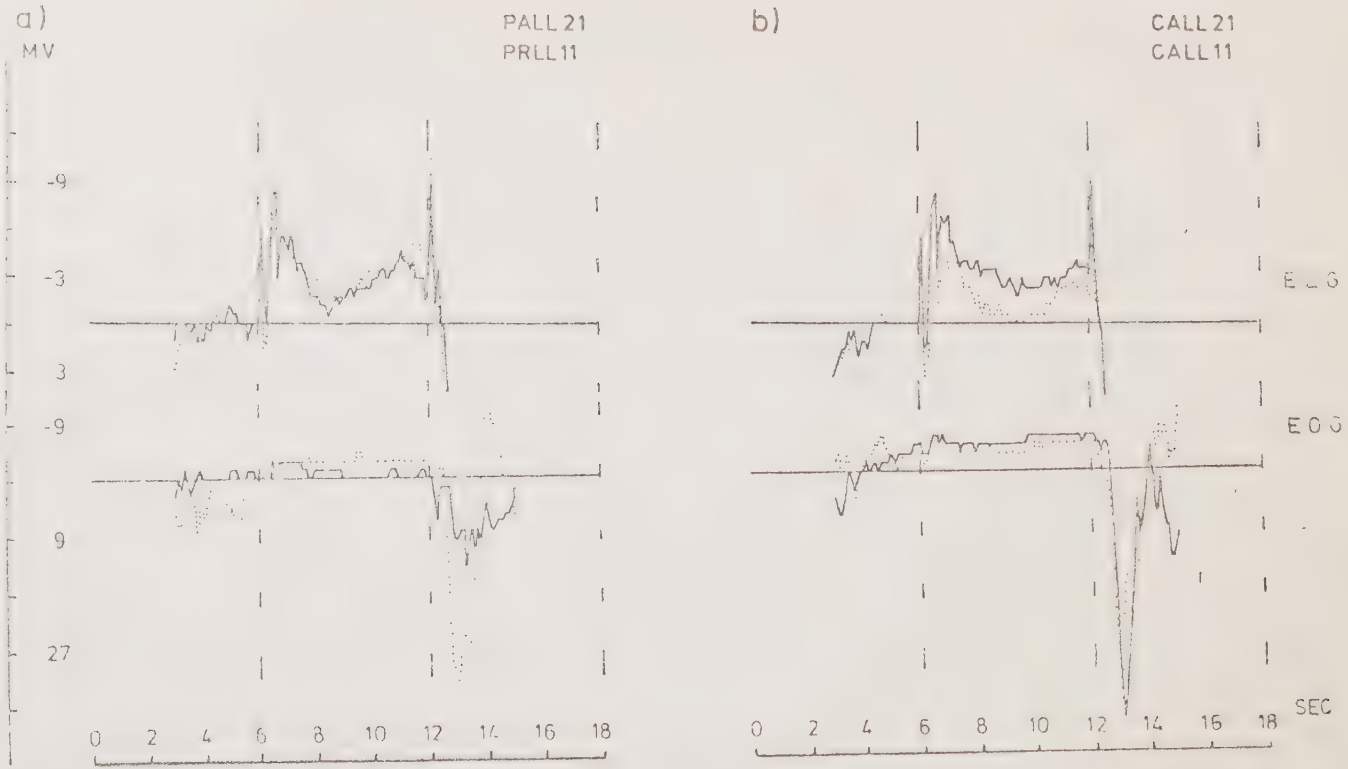
Appendix C4: showing group scores for the behavioural variables in both conditions and sessions.



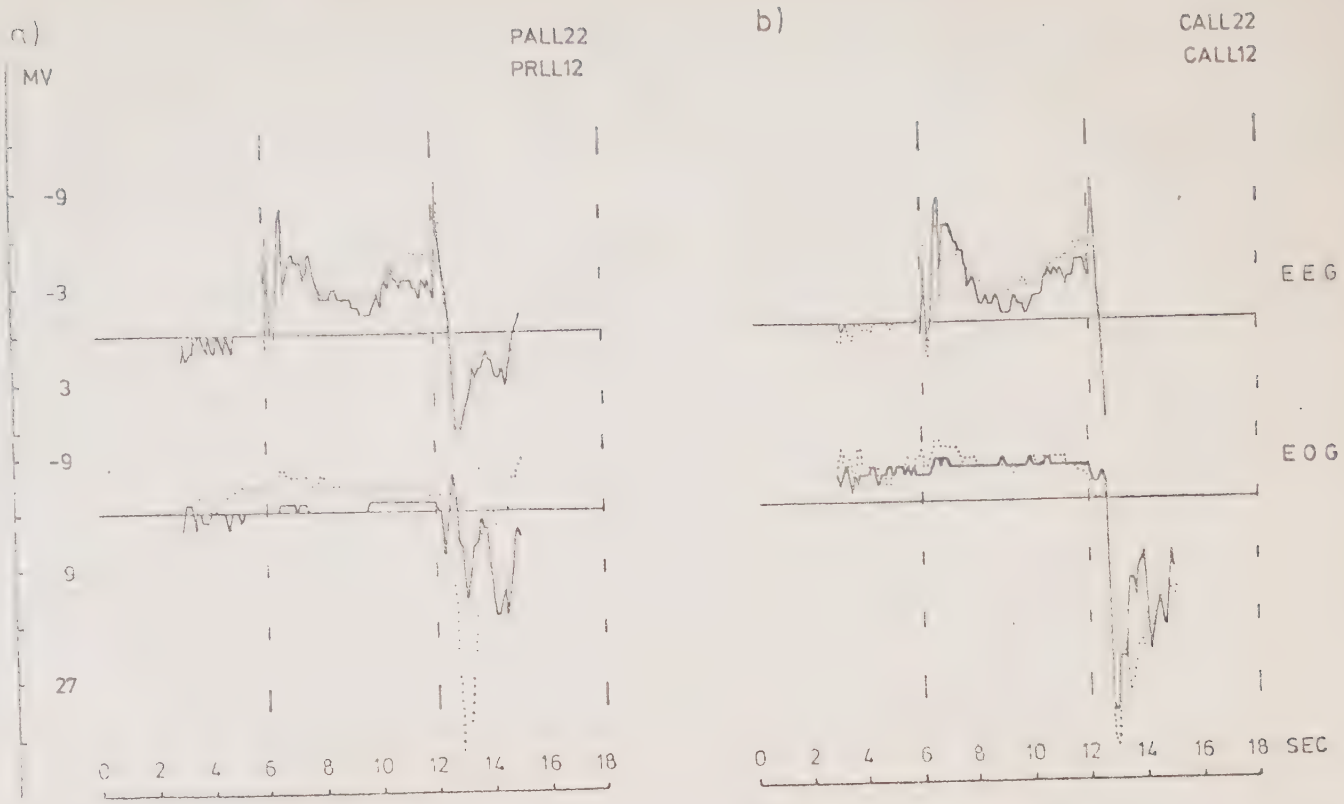
Appendix D1: showing, for reactive depressives (a) and controls (b), the non-incentive (————) and incentive (·····) conditions for session one.



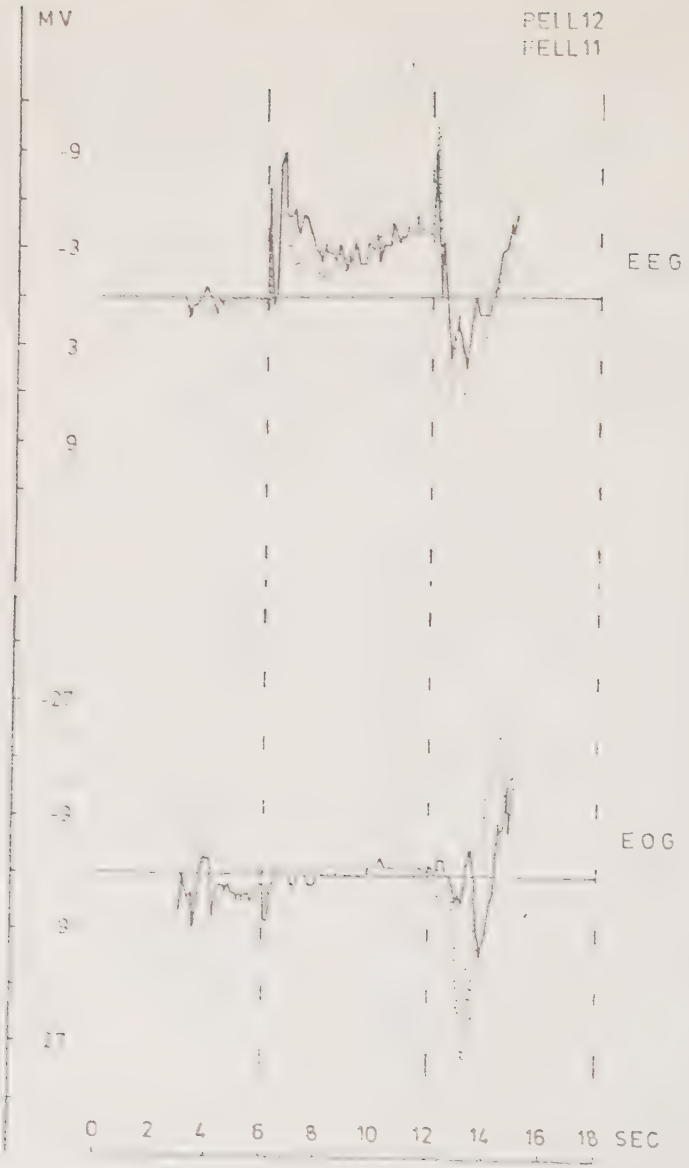
Appendix D2: showing, for reactive depressives (a) and control- (b), the non-incentive (———) and incentive (·····) conditions for session two.



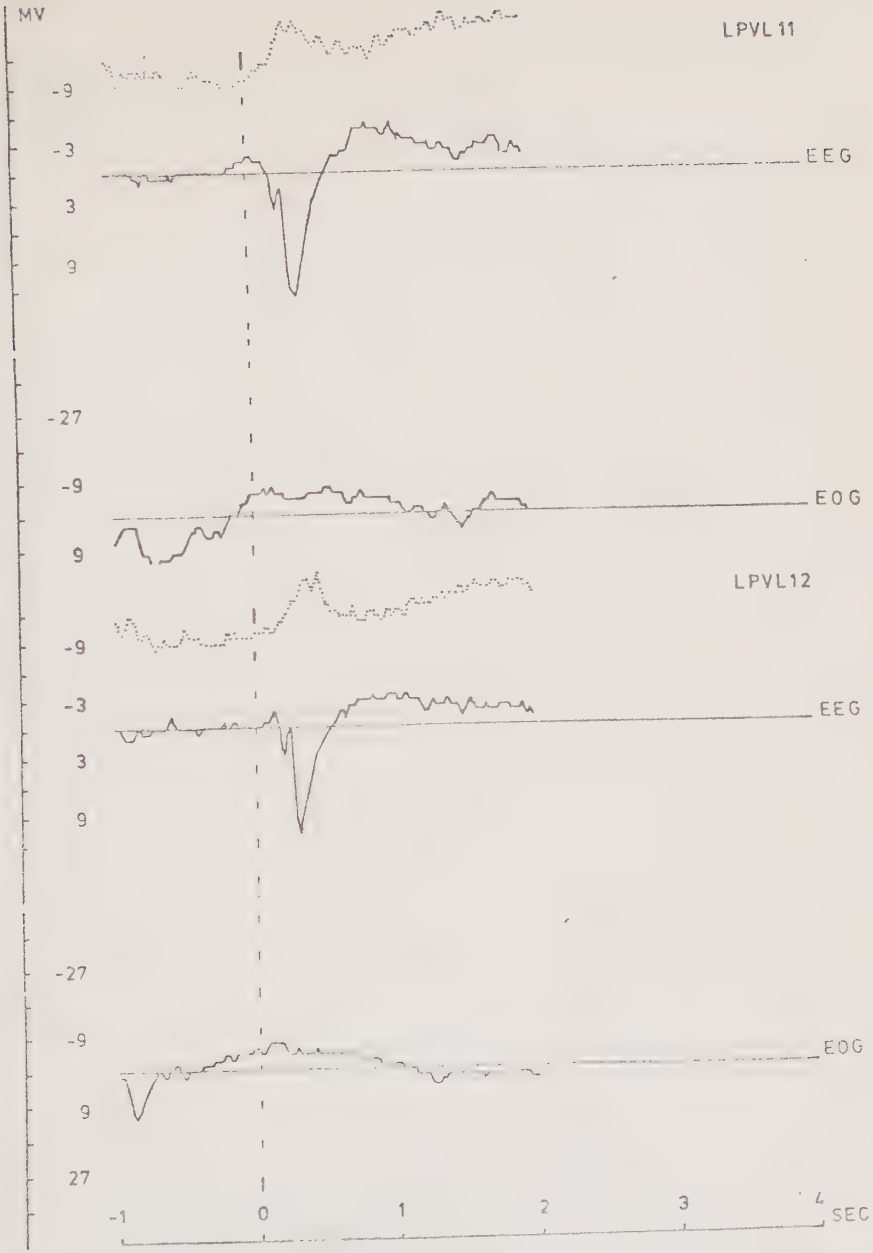
Appendix D3: showing, for reactive depressives (a) and controls (b), the comparison of session one (———) and session two (.....) in the non-incentive condition.



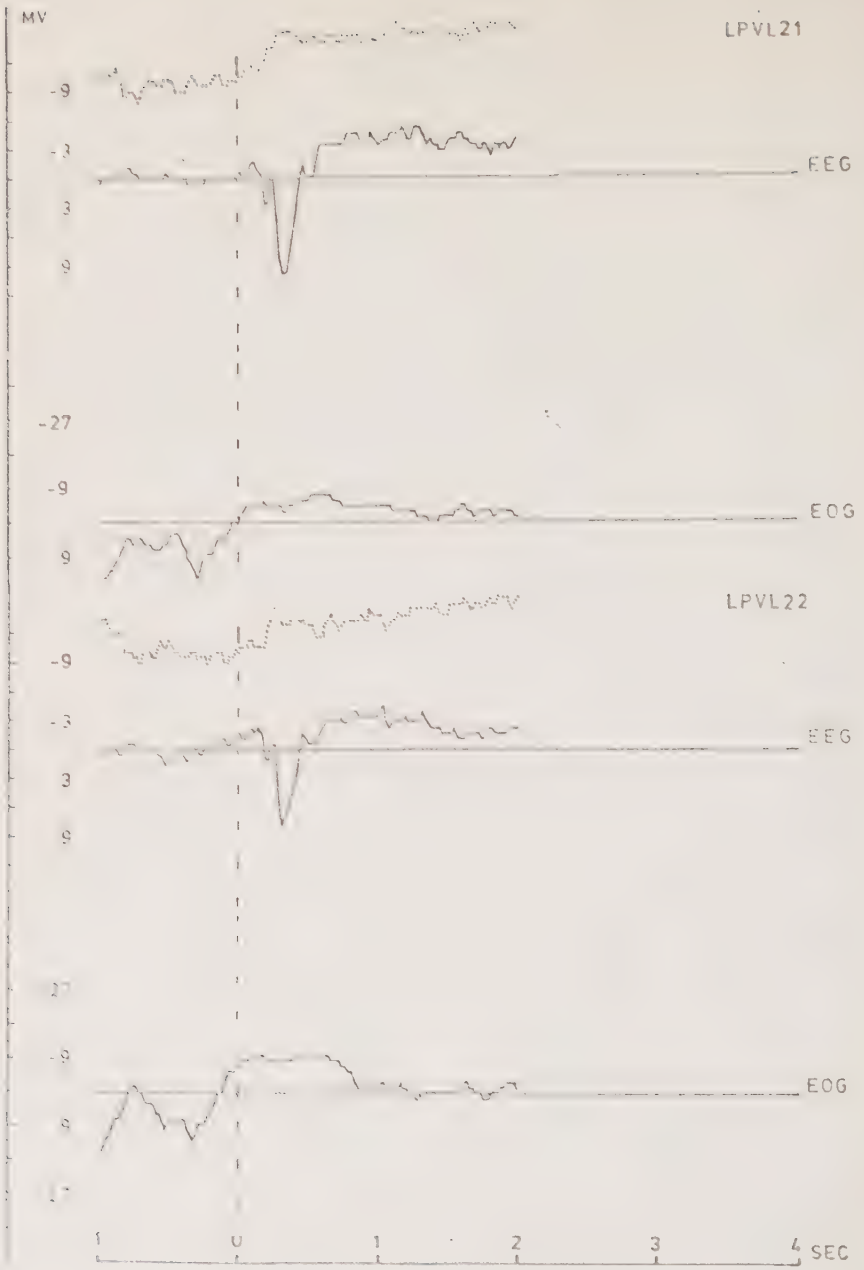
Appendix D4: showing, for reactive depressives (a) and controls (b), the comparison of session one (———) and session two (·····) in the incentive condition.



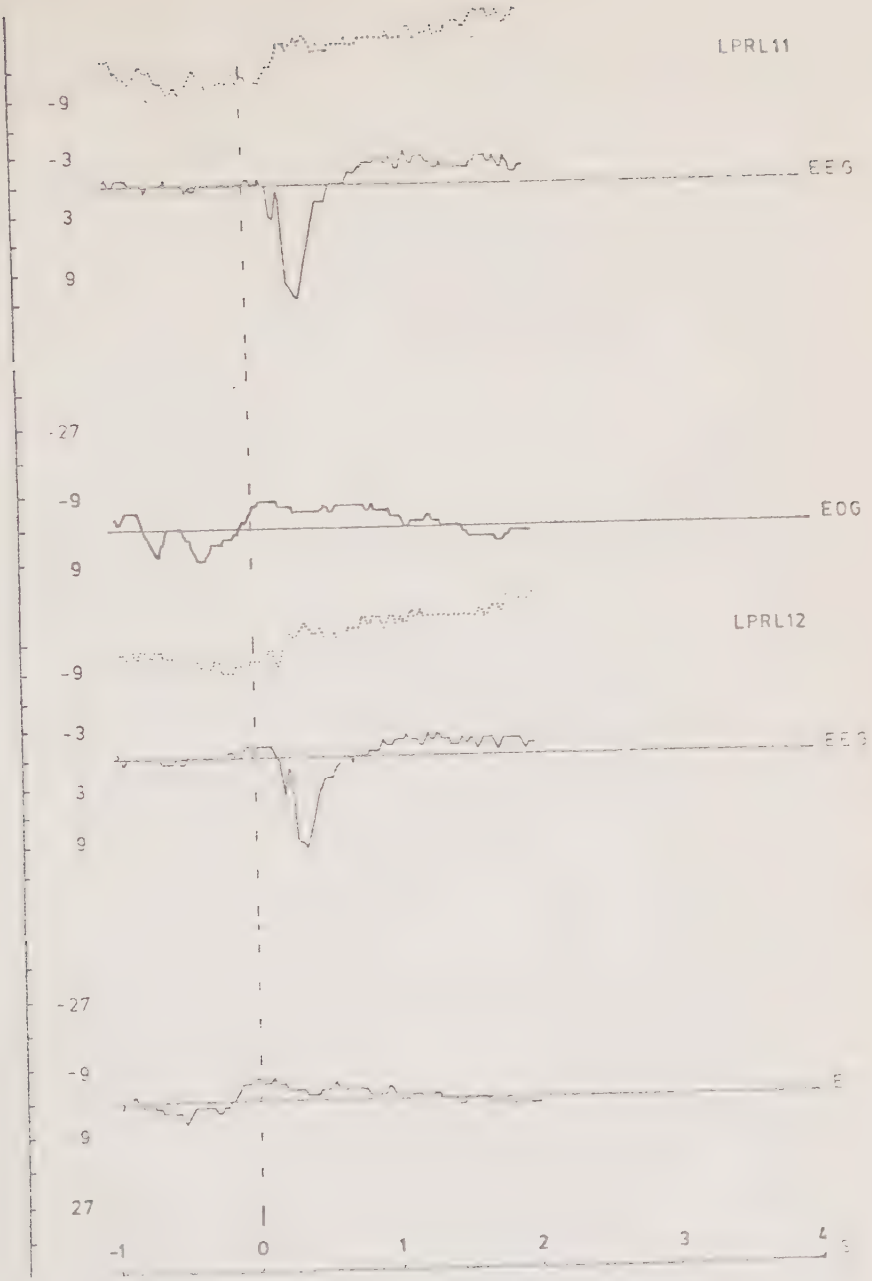
Appendix D5: showing, for endogenous depressives, the comparison of incentive (——) and non-incentive (·····) conditions in session one.



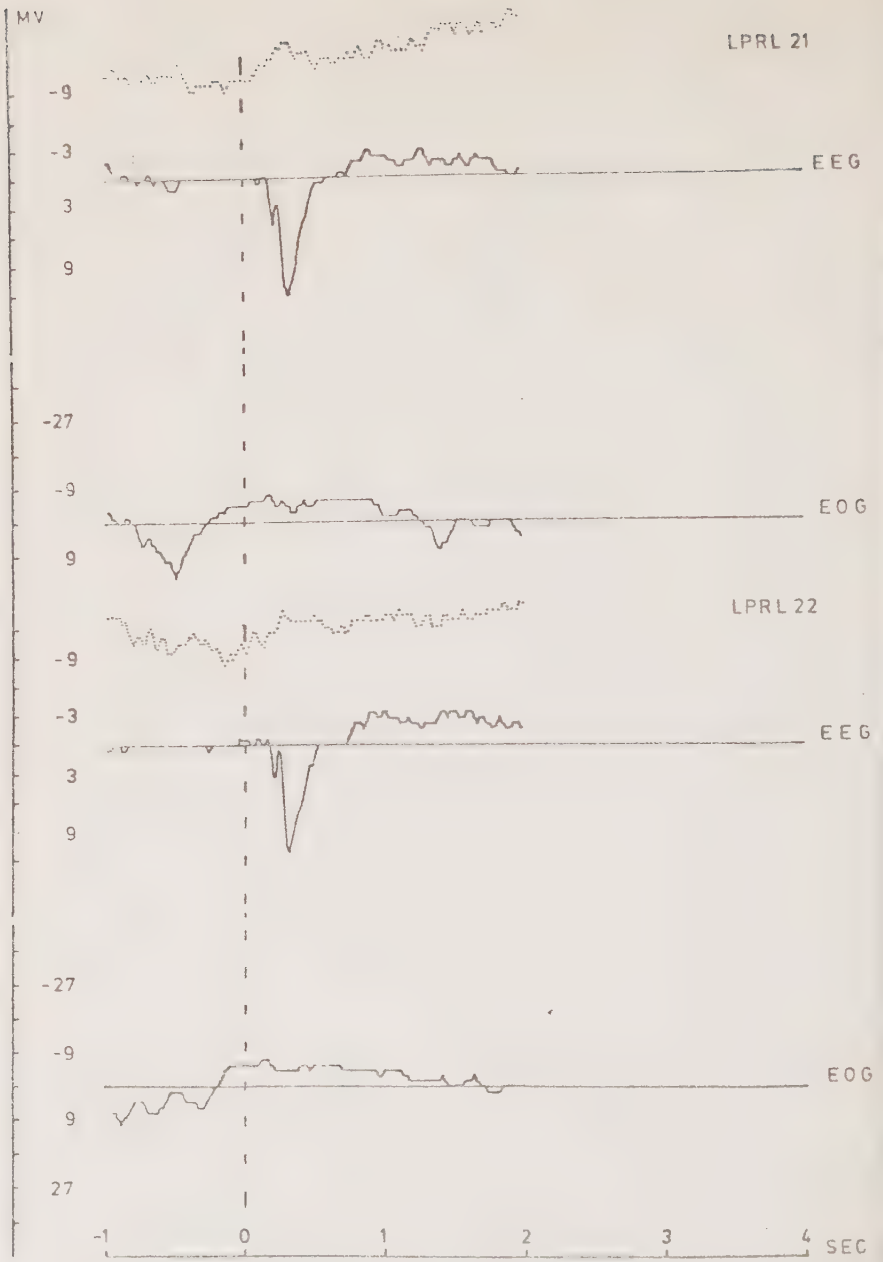
Appendix E:showing, for controls, the averaged waveform (- — —) and the absolute standard deviation (·····) in the non-incentive (above) and incentive (below) coditions of session one. to light onset.



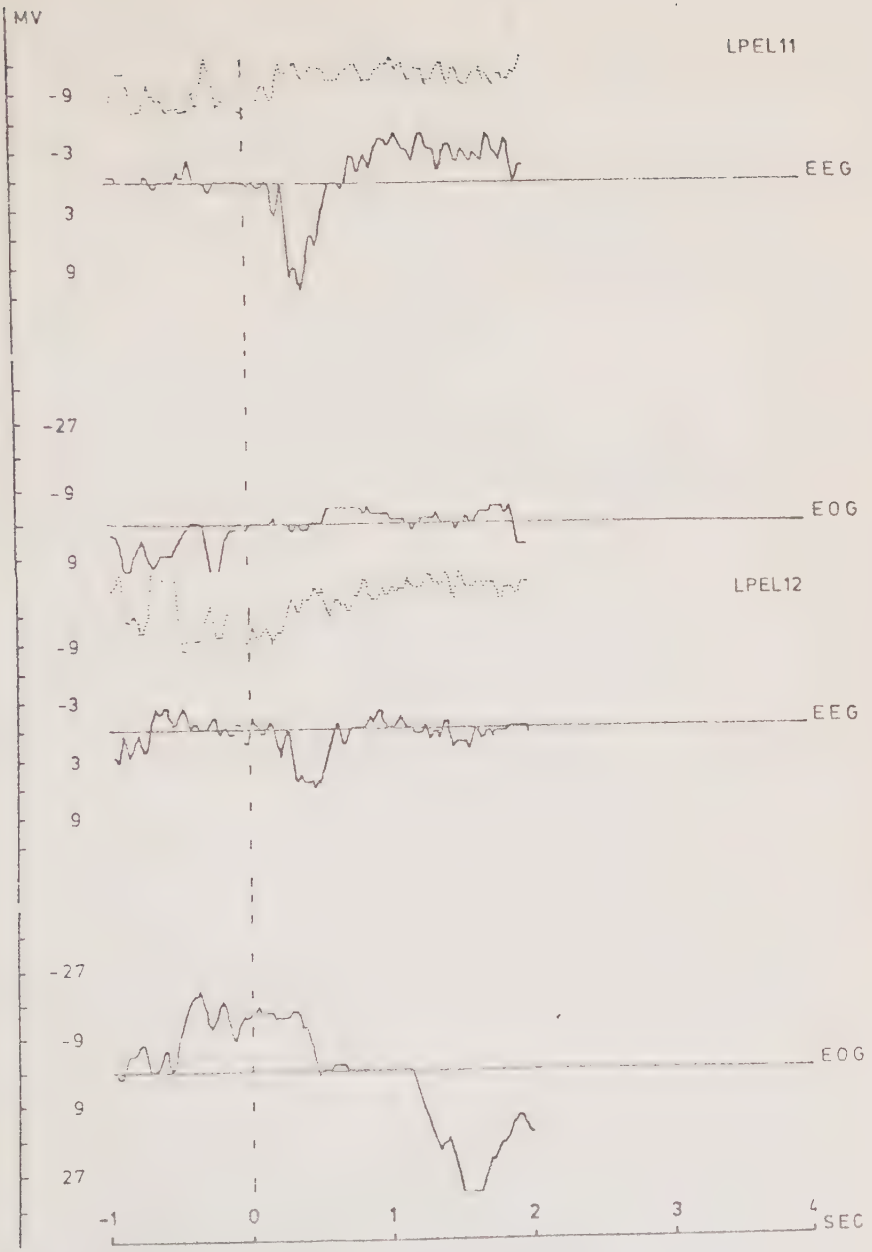
Appendix E: showing, for controls, the averaged waveform (——) and the absolute standard deviation (·····) in the non-incentive (above) and incentive (below) condition of session two to light onset.



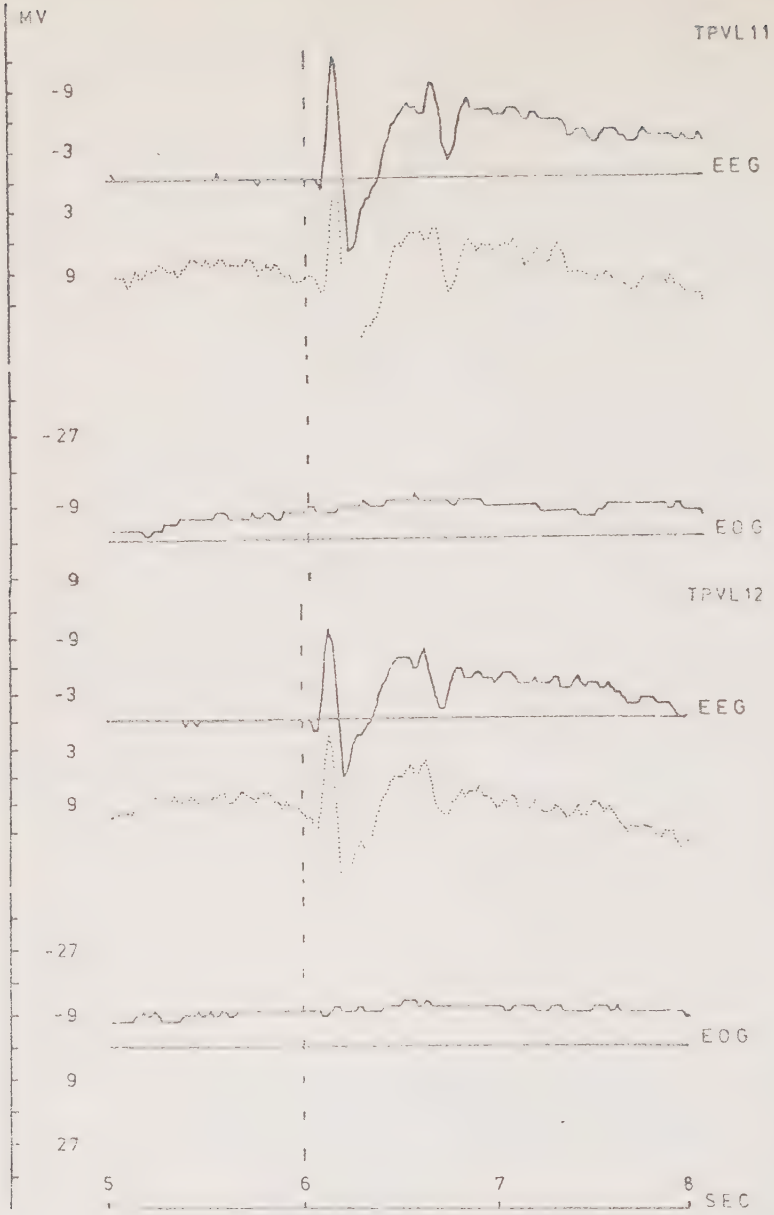
Appendix E: showing, for reactive depressives, the aver-
orms (———) and the absolute standard deviation
the non-incentive (above) and incentive (below)
condition of session one to light onset.



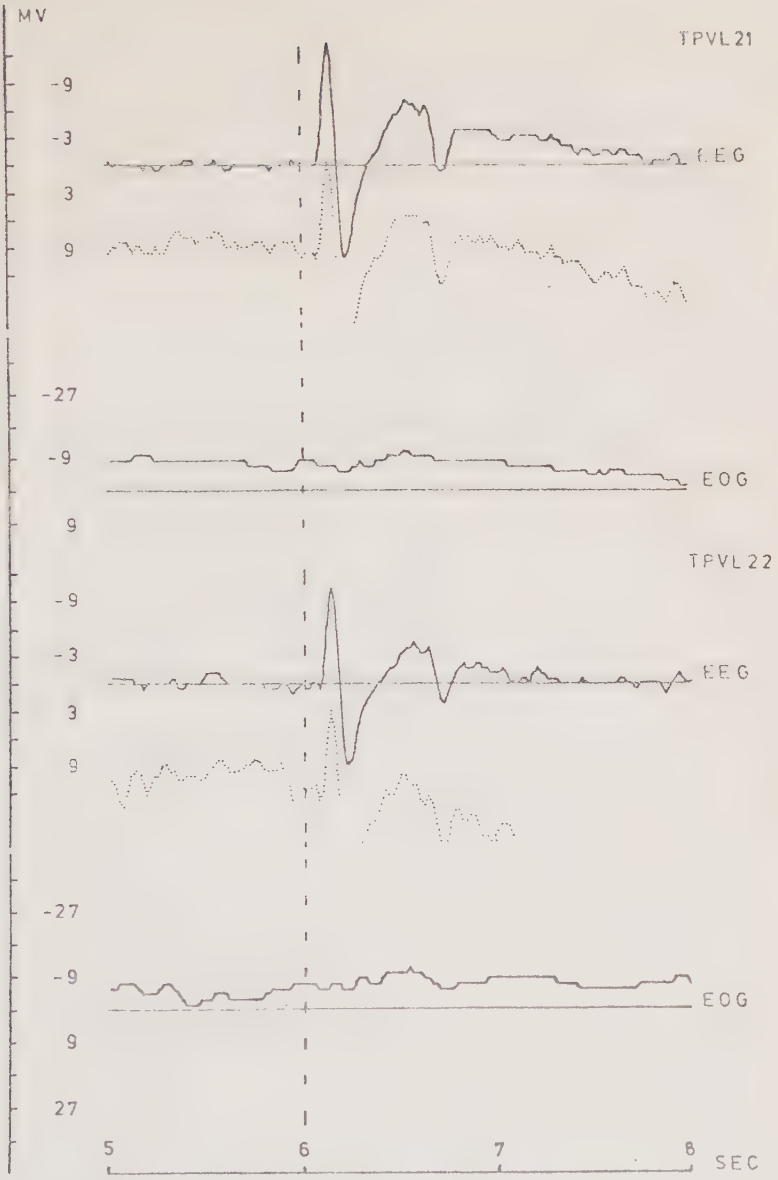
Appendix E: showing, for reactive depressives, the averaged waveform (——) and the absolute standard deviation (·····) in the non-incentive (above) and incentive (below) condition of session two to light onset.



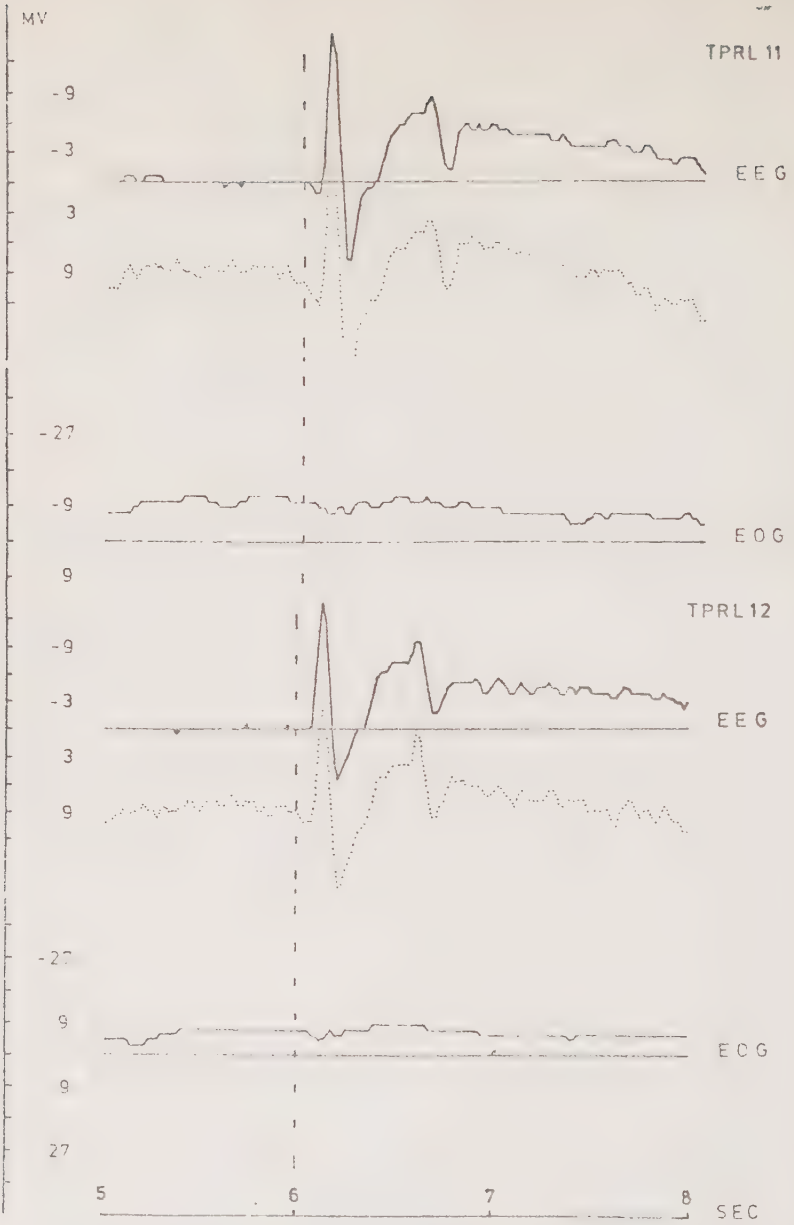
Appendix E: showing, for endogenous depressives, the averaged waveform (——) and the absolute standard deviation (·····) in the non-incentive (above) and incentive (below) condition of session one to light onset.



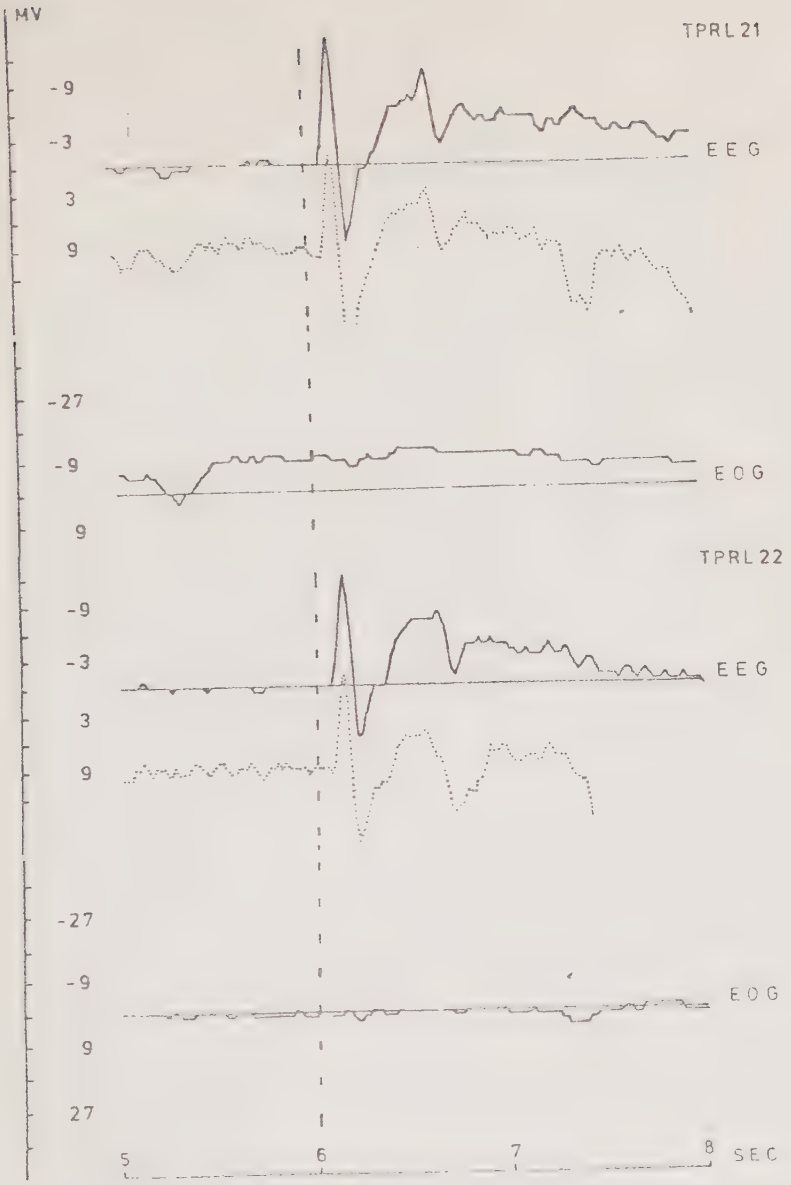
Appendix F: showing, for controls, the averaged waveform (———) and the relative standard deviation (·····) in the [illegible] (above) and incentive (below) condition of session one to tone onset.



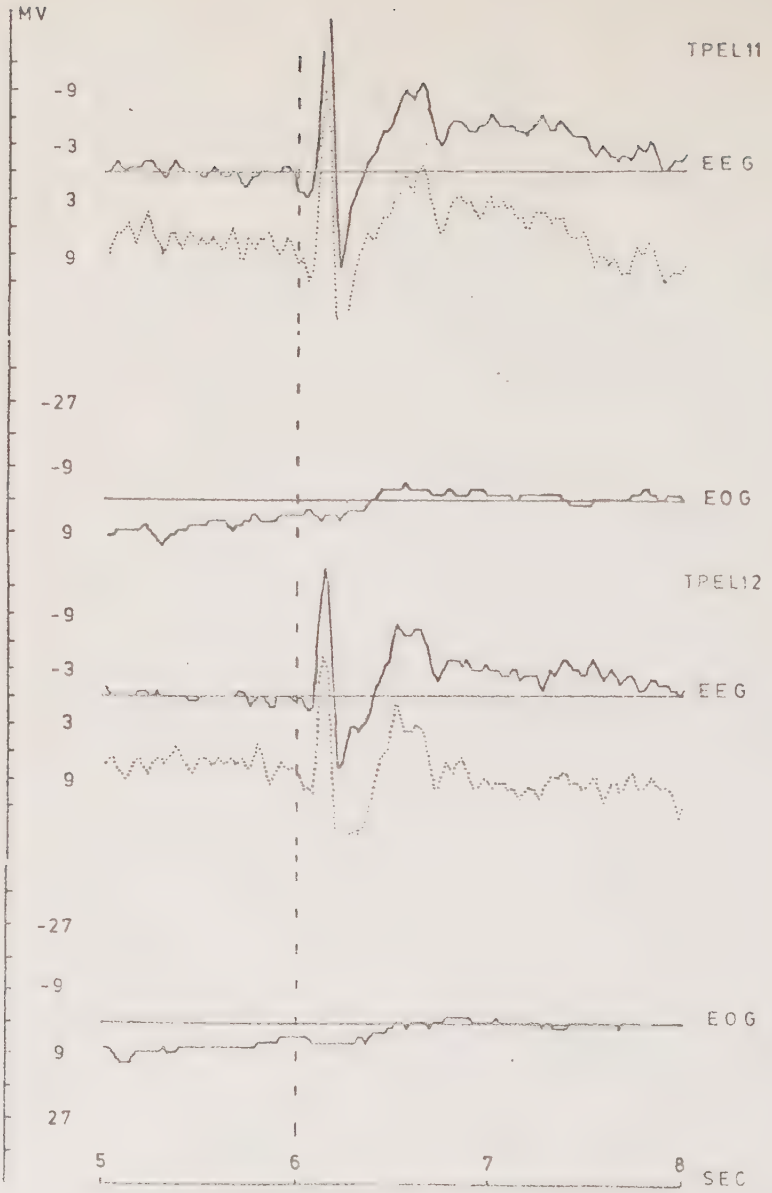
Appendix F: showing, for controls, the averaged waveform (———) and the absolute standard deviation (·····) in the non-incentive (above) and incentive (below) conditions of session two to tone onset.



Appendix F: showing, for reactive depressives, the averaged waveform (——) and the relative standard deviation (·····) in the non-incentive (above) and incentive (below) conditions of session one to tone onset.



Appendix F: showing, for reactive depressives, the averaged waveform (——) and the relative standard deviation (·····) in the non-incentive (above) and incentive (below) condition of session two to tone onset.



Appendix F: showing, for endogenous depressives, the averaged waveform (——) and the relative standard deviation (.....) in the non-incentive (above) and incentive conditions of session one to tone onset.

VARIABLE	U	VARIABLE	U	VARIABLE	U	VARIABLE	U
AMP111	149.0 (0.25)	AMP112	157.5 (0.36)	AMP121	156.0 (0.34)	AMP122	163.5 (0.45)
AMP211	187.5 (0.94)	AMP212	179.0 (0.76)	AMP221	172.0 (0.61)	AMP222	163.5 (0.45)
LAT111	157.5 (0.36)	LAT211	144.0 (0.19)	LAT121	179.0 (0.77)	LAT122	145.5 (0.21)
LAT211	180.5 (0.79)	LAT212	178.5 (0.74)	LAT221	136.5 (0.13)	LAT222	173.5 (0.64)
AR111	145.0 (0.21)	AR112	176.5 (0.70)	AR121	148.5 (0.24)	AR122	171.5 (0.60)
AR211	189.0 (0.98)	AR212	161.5 (0.42)	AR221	176.5 (0.70)	AR222	155.0 (0.32)
TNP111	129.5 (0.30)	TNP112	124.5 (0.23)	TNP121	127.0 (0.74)	TNP122	144.5 (0.78)
TNP211	148.5 (0.67)	TNP212	144.0 (0.57)	TNP221	135.0 (0.97)	TNP222	152.5 (0.99)
TLAT11	150.0 (0.69)	TLAT12	148.0 (0.64)	TLAT21	118.0 (0.49)	TLAT22	147.5 (0.85)
TMAX11	152.0 (0.75)	TMAX12	142.0 (0.53)	TMAX21	118.5 (0.52)	TMAX22	114.0 (0.20)
TN11	133.5 (0.11)	TN12	115.0 (0.03)	TN21	189.5 (0.99)	TN22	144.5 (0.20)
LNP111	168.0 (0.93)	LNP112	164.0 (0.83)	LNP121	131.0 (0.64)	LNP122	128.5 (0.29)
LNP211	148.0 (0.48)	LNP212	161.0 (0.76)	LNP221	119.5 (0.39)	LNP222	146.0 (0.62)
LLAT11	155.5 (0.63)	LLAT12	162.5 (0.79)	LLAT21	131.5 (0.65)	LLAT22	126.5 (0.26)
LMAX11	169.5 (0.96)	LMAX12	160.5 (0.75)	LMAX21	126.5 (0.53)	LMAX22	105.5 (0.07)
LN11	172.0 (0.61)	LN12	186.0 (0.91)	LN21	126.0 (0.99)	LN22	172.0 (0.20)

Appendix G1: showing the Mann-Whitney statistic to test for differences between reactive depressives and controls for the CNV, and ERP, to both tone and light onset, measures; the associated exact 2-tailed probability is shown in parenteses. Standard deviation and behavioural measures are reproduced below.

A.				B.			
VARIABLE	U	VARIABLE	U	VARIABLE	U	VARIABLE	U
AMP111	21.0	AMP112	24.0	AMP111	18.0	AMP112	19.5
	(0.65)		(0.94)		(0.40)		(0.51)
AMP211	22.5	AMP212	17.0	AMP211	16.5	AMP212	21.0
	(0.79)		(0.33)		(0.30)		(0.65)
LAT111	20.0	LAT112	11.5	LAT111	20.5	LAT112	21.0
	(0.56)		(0.09)		(0.60)		(0.65)
LAT211	11.5	LAT212	20.5	LAT211	15.5	LAT212	21.0
	(0.09)		(0.60)		(0.24)		(0.65)
AR111	24.5	AR112	21.0	AR111	22.0	AR112	19.5
	(1.00)		(0.65)		(0.74)		(0.52)
AR211	24.0	AR212	23.0	AR211	22.5	AR212	22.5
	(0.94)		(0.84)		(0.79)		(0.79)
TA11	23.0	TA12	20.0	TA11	20.0	TA12	21.5
	(0.84)		(0.56)		(0.56)		(0.70)
TNP111	9.0	TNP112	11.0	TNP111	19.5	TNP112	11.5
	(0.04)		(0.08)		(0.82)		(0.17)
TNP211	13.0	TNP212	12.0	TNP211	16.0	TNP212	17.0
	(0.14)		(0.11)		(0.47)		(0.56)
TLAT11	10.0	TLAT12	18.0	TLAT11	6.0	TLAT12	11.5
	(0.05)		(0.36)		(0.02)		(0.15)
TMAX11	21.0	TMAX12	22.5	TMAX11	17.5	TMAX12	17.5
	(0.65)		(0.79)		(0.61)		(0.61)
TN11	3.0	TN12	9.0	TN11	12.0	TN12	24.0
	(0.006)		(0.05)		(0.11)		(.95)
LNP111	24.0	LNP112	22.5	LNP111	7.5	LNP112	15.0
	(0.94)		(0.79)		(0.02)		(0.22)
LNP211	19.0	LNP212	22.5	LNP211	16.5	LNP212	21.0
	(0.48)		(0.79)		(0.30)		(0.65)
LLAT11	21.0	LLAT12	18.0	LLAT11	23.0	LLAT12	21.5
	(0.64)		(0.39)		(0.84)		(0.69)
LMAX11	21.0	LMAX12	24.0	LMAX11	19.0	LMAX12	23.0
	(0.65)		(0.94)		(0.48)		(0.84)
LN11	22.0	LN12	24.0	LN11	13.0	LN12	14.0
	(0.74)		(0.95)		(0.14)		(0.18)

Appendix G2: showing the Mann-Whitney statistic for group differences between endogenous depressives and reactive (B) and controls (A) for the CNV and ERP, to both tone and light onset. Two-tailed probabiliy is reported in parentheses. Other measures are reported below.

VARIABLE	U	VARIABLE	U	VARIABLE	U	VARIABLE	U
EEG111	174.0	EEG112	169.0	EEG121	180.5	EEG122	130.0
	(0.65)		(0.55)		(1.00)		(0.09)
EEG211	186.5	EEG212	168.0	EEG221	171.5	EEG222	188.5
	(0.92)		(0.45)		(0.79)		(0.96)
EEG311	177.0	EEG312	173.5	EEG321	179.5	EEG322	189.0
	(0.71)		(0.64)		(0.78)		(0.98)
SDL11	118.0	SDL12	137.0	SDL21	129.0	SDL22	103.0
	(0.26)		(0.81)		(0.80)		(0.25)
SDT11	145.5	SDT12	157.5	SDT21	140.5	SDT22	132.0
	(0.82)		(0.68)		(0.68)		(0.49)
AVRT11	145.5	AVRT12	137.0	AVRT21	144.5	AVRT22	152.5
	(0.21)		(0.14)		(0.20)		(0.29)
ZUF11	162.0			ZUF21	141.5		
	(0.43)				(0.37)		
BF11	38.0	BK11	0.0	BF21	54.5		
	(0.0001)		(0.0000)		(0.0000)		

A.

VARIABLE	U	VARIABLE	U	VARIABLE	U	VARIABLE	U
EEG111	8.0	EEG112	23.5	EEG111	8.0	EEG112	12.5
	(0.03)		(0.89)		(0.03)		(0.12)
EEG211	14.0	EEG212	20.5	EEG111	14.0	EEG212	13.5
	(0.17)		(0.60)		(0.17)		(0.15)
EEG311	20.0	EEG312	23.5	EEG311	21.5	EEG312	15.5
	(0.56)		(0.89)		(0.70)		(0.24)
SDL11	13.0	SDL12	20.5	SDL11	8.5	SDL12	20.5
	(0.25)		(0.94)		(0.04)		(0.61)
SDT11	23.0	SDT12	22.5	SDT11	21.5	SDT12	21.5
	(0.85)		(0.80)		(0.70)		(0.70)
AVRT11	16.0	AVRT12	13.0	AVRT11	19.0	AVRT12	19.5
	(0.28)		(0.14)		(0.48)		(0.52)
ZUF11	20.5			ZUF11	16.0		
	(0.94)				(0.47)		
BF11	8.5	BK11	0.0	BF21	15.5	BK21	2.0
	(0.07)		(0.007)		(0.69)		(0.05)

Appendices G1 and 2 (continued): showing, for standard deviation and behavioural measures, the Mann-Whitney U-statistic for controls and reactive depressives (above), and (below) for endogenous and reactive depressives (B) and controls (A); exact two-tailed probabilities are shown in parentheses.

	NON-INCENTIVE COND.			INCENTIVE CONDITION		
VARIABLE	AVRT	Bf	Bk	AVRT	Bf	Bk
AMP1	0.134	-0.179	-0.417	0.103	-0.182	-0.144
	(0.29)	(0.23)	(0.04)	(0.34)	(0.23)	(0.28)
AMP2	0.035	-0.047	-0.117	0.031	-0.058	-0.029
	(0.44)	(0.42)	(0.32)	(0.45)	(0.41)	(0.45)
LAT1	-0.091	0.015	-0.308	0.071	-0.641	-0.450
	(0.34)	(0.48)	(0.10)	(0.39)	(0.002)	(0.03)
LAT2	0.026	0.380	0.009	0.131	0.116	0.191
	(0.46)	(0.05)	(0.48)	(0.30)	(0.32)	(0.22)
AR1	-0.084	-0.042	-0.281	-0.096	-0.241	-0.163
	(0.37)	(0.43)	(0.12)	(0.35)	(0.16)	(0.25)
AR2	0.061	-0.121	-0.132	-0.021	-0.093	0.024
	(0.40)	(0.31)	(0.29)	(0.47)	(0.35)	(0.46)
TA	0.012	0.073	-0.115	0.029	-0.141	-0.045
	(0.48)	(0.38)	(0.32)	(0.45)	(0.28)	(0.43)
TNP1	-0.361	-0.076	0.014	-0.585	-0.024	-0.033
	(0.08)	(0.38)	(0.48)	(0.005)	(0.46)	(0.45)
TNP2	-0.367	-0.158	-0.174	-0.557	-0.123	-0.103
	(0.07)	(0.27)	(0.24)	(0.008)	(0.31)	(0.34)
TLAT	0.069	0.021	0.137	0.087	0.414	0.329
	(0.39)	(0.47)	(0.29)	(0.36)	(0.04)	(0.09)
TMAX	-0.244	0.063	0.081	-0.537	0.057	0.074
	(0.16)	(0.40)	(0.37)	(0.01)	(0.41)	(0.38)
TN	-0.300	-0.025	0.027	-0.321	0.083	-0.060
	(0.11)	(0.46)	(0.46)	(0.09)	(0.37)	(0.40)
LNP1	0.075	0.072	-0.010	0.078	-0.093	0.232
	(0.38)	(0.39)	(0.48)	(0.38)	(0.36)	(0.18)
LNP2	0.095	-0.190	-0.386	-0.055	-0.097	-0.248
	(0.35)	(0.22)	(0.06)	(0.41)	(0.35)	(0.16)
LLAT	0.313	0.054	0.033	-0.025	0.233	0.024
	(0.10)	(0.41)	(0.45)	(0.46)	(0.18)	(0.46)
LMAX	-0.001	0.123	-0.063	0.208	0.216	-0.171
	(0.50)	(0.30)	(0.40)	(0.20)	(0.19)	(0.25)
LN	0.176	-0.134	0.074	-0.142	-0.490	-0.167
	(0.23)	(0.29)	(0.38)	(0.28)	(0.02)	(0.25)

Appendix H1): showing the Pearson correlation coeffic- , with the exact probability in parentheses, between the CNV and ERP variables and the behavioural for controls in both conditions of session one.

	NON-INCENTIVE COND.			INCENTIVE CONDITION		
VARIABLE	AVRT	Bf	Bk	AVRT	Bf	Bk
AMP1	-0.052	-0.017	0.477	0.278	-0.221	-0.021
	(0.41)	(0.47)	(0.02)	(0.12)	(0.19)	(0.47)
AMP2	-0.182	0.301	0.373	0.182	-0.150	-0.300
	(0.22)	(0.11)	(0.06)	(0.22)	(0.28)	(0.11)
LAT1	0.145	0.255	-0.122	0.032	-0.205	-0.505
	(0.27)	(0.15)	(0.31)	(0.45)	(0.21)	(0.01)
LAT2	0.004	-0.108	0.151	-0.178	-0.373	-0.400
	(0.49)	(0.33)	(0.26)	(0.23)	(0.06)	(0.04)
AR1	-0.100	-0.013	0.516	0.417	-0.252	-0.171
	(0.34)	(0.48)	(0.01)	(0.03)	(0.16)	(0.47)
AR2	-0.204	0.178	0.255	0.069	-0.029	-0.242
	(0.19)	(0.24)	(0.14)	(0.39)	(0.45)	(0.16)
TA	-0.200	0.122	0.528	0.275	-0.120	-0.176
	(0.20)	(0.31)	(0.01)	(0.12)	(0.32)	(0.23)
TNP1	0.068	0.028	-0.046	-0.133	-0.006	0.042
	(0.40)	(0.46)	(0.43)	(0.30)	(0.49)	(0.44)
TNP2	0.218	0.043	0.298	0.283	0.043	0.455
	(0.19)	(0.44)	(0.12)	(0.13)	(0.44)	(0.03)
TLAT	0.239	0.001	0.152	0.493	-0.303	-0.014
	(0.17)	(0.50)	(0.28)	(0.02)	(0.13)	(0.48)
TMAX	0.184	0.218	0.262	0.250	0.142	0.556
	(0.23)	(0.21)	(0.15)	(0.16)	(0.30)	(0.01)
TN	-0.220	-0.107	-0.141	-0.488	-0.092	-0.368
	(0.18)	(0.34)	(0.28)	(0.01)	(0.36)	(0.06)
LNP1	0.247	-0.399	-0.034	0.263	0.013	0.218
	(0.15)	(0.06)	(0.45)	(0.14)	(0.48)	(0.19)
LNP2	0.246	0.031	0.241	0.259	0.042	0.269
	(0.15)	(0.45)	(0.17)	(0.14)	(0.44)	(0.14)
LLAT	0.040	-0.307	0.040	0.324	-0.243	-0.003
	(0.43)	(0.12)	(0.40)	(0.08)	(0.17)	(0.50)
LMAX	0.298	0.070	0.383	0.298	0.086	0.296
	(0.11)	(0.39)	(0.09)	(0.11)	(0.37)	(0.12)
LN	-0.067	-0.574	-0.472	-0.045	-0.080	-0.060
	(0.39)	(0.006)	(0.02)	(0.42)	(0.38)	(0.40)

Appendix H2): showing the Pearson correlation coefficient, with the exact probabilities in parenteses, between the CNV and ERP variables and the behavioural for reactive depressives in both conditions of session one.

	NON-INCENTIVE COND.		INCENTIVE CONDITION	
VARIABLE	AVRT	Bf	AVRT	BF
AMP1	-0.083	-0.335	0.146	-0.062
	(0.37)	(0.09)	(0.27)	(0.40)
AMP2	-0.273	-0.083	0.169	0.095
	(0.13)	(0.37)	(0.24)	(0.35)
LAT1	0.009	0.438	0.259	0.420
	(0.48)	(0.03)	(0.14)	(0.04)
LAT2	-0.179	0.261	-0.341	-0.310
	(0.23)	(0.15)	(0.08)	(0.10)
AR1	-0.226	-0.263	0.427	0.348
	(0.18)	(0.14)	(0.03)	(0.08)
AR2	-0.387	-0.101	0.290	0.151
	(0.05)	(0.34)	(0.11)	(0.27)
TA	-0.342	-0.184	0.368	0.225
	(0.08)	(0.23)	(0.06)	(0.18)
TNP1	-0.285	-0.170	0.308	-0.153
	(0.13)	(0.26)	(0.11)	(0.29)
TNP2	-0.309	-0.168	-0.441	-0.274
	(0.11)	(0.27)	(0.04)	(0.15)
TLAT	0.205	0.188	-0.132	0.366
	(0.21)	(0.24)	(0.31)	(0.08)
TMAX	0.299	-0.049	-0.333	-0.057
	(0.12)	(0.43)	(0.10)	(0.42)
TN	-0.042	-0.286	-0.140	-0.265
	(0.43)	(0.12)	(0.28)	(0.14)
LNP1	0.510	-0.060	0.170	-0.256
	(0.42)	(0.41)	(0.26)	(0.17)
LNP2	0.043	-0.280	-0.078	-0.134
	(0.43)	(0.15)	(0.38)	(0.31)
LLAT	0.499	-0.456	0.343	0.391
	(0.02)	(0.32)	(0.09)	(0.07)
LMAX	0.150	-0.184	-0.007	-0.130
	(0.28)	(0.25)	(0.49)	(0.32)
LN	-0.136	0.147	0.169	-0.128
	(0.29)	(0.28)	(0.24)	(0.31)

Appendix H3): showing the Pearson correlation coefficient, with the exact probability in parentheses, between the CNV and ERP variables and the behavioural for controls in both conditions of session two.

	NON-INCENTIVE COND.			INCENTIVE CONDITION		
VARIABLE	AVRT	Bf	Bk	AVRT	Bf	Bk
AMP1	-0.196	-0.006	-0.241	-0.098	-0.103	0.105
	(0.20)	(0.49)	(0.17)	(0.34)	(0.48)	(0.34)
AMP2	-0.240	-0.357	-0.363	-0.193	-0.415	-0.265
	(0.15)	(0.08)	(0.08)	(0.21)	(0.05)	(0.15)
LAT1	-0.117	-0.218	-0.066	0.178	0.127	0.374
	(0.31)	(0.20)	(0.40)	(0.23)	(0.31)	(0.07)
LAT2	0.020	-0.091	0.146	-0.170	0.160	0.082
	(0.47)	(0.36)	(0.29)	(0.24)	(0.27)	(0.38)
AR1	-0.123	0.032	-0.060	-0.034	-0.095	0.076
	(0.30)	(0.45)	(0.41)	(0.44)	(0.36)	(0.39)
AR2	-0.138	-0.260	-0.224	-0.133	-0.388	-0.207
	(0.28)	(0.16)	(0.19)	(0.29)	(0.06)	(0.21)
TA	-0.115	-0.110	-0.115	-0.052	-0.505	-0.303
	(0.31)	(0.34)	(0.33)	(0.41)	(0.02)	(0.12)
TNP1	-0.339	-0.561	0.516	-0.420	-0.393	-0.539
	(0.30)	(0.02)	(0.04)	(0.04)	(0.07)	(0.02)
TNP2	-0.160	0.019	-0.105	-0.051	-0.020	-0.163
	(0.28)	(0.47)	(0.37)	(0.42)	(0.47)	(0.28)
TLAT	0.515	-0.131	-0.253	0.090	-0.415	-0.252
	(0.02)	(0.33)	(0.20)	(0.36)	(0.06)	(0.18)
TMAX	0.099	0.149	0.046	-0.196	0.018	-0.190
	(0.36)	(0.31)	(0.44)	(0.22)	(0.47)	(0.25)
TN	-0.069	-0.266	-0.211	-0.311	-0.242	-0.191
	(0.39)	(0.15)	(0.21)	(0.09)	(0.17)	(0.23)
LNP1	0.212	0.159	0.257	0.452	0.108	-0.037
	(0.21)	(0.29)	(0.19)	(0.03)	(0.35)	(0.44)
LNP2	0.022	0.120	0.187	0.232	0.196	0.154
	(0.47)	(0.34)	(0.26)	(0.17)	(0.23)	(0.28)
LLAT	0.407	0.198	0.231	0.174	-0.003	0.093
	(0.05)	(0.25)	(0.21)	(0.24)	(0.50)	(0.37)
LMAX	0.388	0.601	0.472	0.489	0.404	0.128
	(0.06)	(0.01)	(0.04)	(0.02)	(0.06)	(0.32)
LN	-0.146	-0.380	-0.145	-0.071	-0.374	-0.181
	(0.27)	(0.07)	(0.29)	(0.38)	(0.07)	(0.24)

Appendix H4): showing the Pearson correlation coefficient, with the exact probability in parentheses, between the CNV and ERP variables and the behavioural for reactive depressives in both conditions of session two.

	GROUP					
	CONTROL.		REACT.DEP.		ENDOG.DEP.	
VARIABLE	BF1	BK1	BF1	BK1	BF1	BK1
AVRT11	-0.103	-0.248	-0.264	0.075	0.214	0.30
	(0.34)	(0.15)	(0.14)	(0.38)	(0.32)	(0.31)
AVRT12	-0.076	-0.261	-0.314	0.211	0.250	0.20
	(0.38)	(0.14)	(0.10)	(0.19)	(0.24)	(0.37)
BF1		0.652		0.646		0.800
		(0.001)		(0.002)		(0.05)

	BF2	BK2	BF2	BK2
AVRT21	-0.085		0.335	0.430
	(0.37)		(0.09)	(0.04)
AVRT22	0.038		0.280	0.296
	(0.44)		(0.14)	(0.12)
BF				0.816
				(0.001)

Appendix I1): showing the intercorrelation for the behavioural variables within the two sessions for both reactive and endogenous depressives and for controls: exact two-tailed probability is shown in parentheses.

CONTROLS

	BF2		AVRT21		AVRT22	
BF1	r=0.561	AVRT11	r=0.643	AVRT12	r=0.725	
	(0.007)		(0.001)		(0.001)	
BF1	Z=-0.806	AVRT11	Z=-0.644	AVRT12	Z=-1.67	
	(0.42)		(0.52)		(0.09)	

REACTIVE DEPRESSIVES.

	BF2		BK2		AVRT21		AVRT22
BF1	r=0.688	BK1	r=0.532	AVRT11	r=0.443	AVRT12	r=0.701
	(0.001)		(0.01)		(0.02)		(0.001)
BF1	Z=-0.659	BK1	Z=-3.15	AVRT11	Z=-0.989	AVRT12	Z=-0.261
	(0.51)		(0.002)		(0.32)		(0.79)

Appendix I2): showing for controls (above) and reactive depressives (below) the intra-session Pearson correlation coefficients (r) and the Wilcoxon statistic (Z): exact two-tailed probabilities are shown in parenteses.

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